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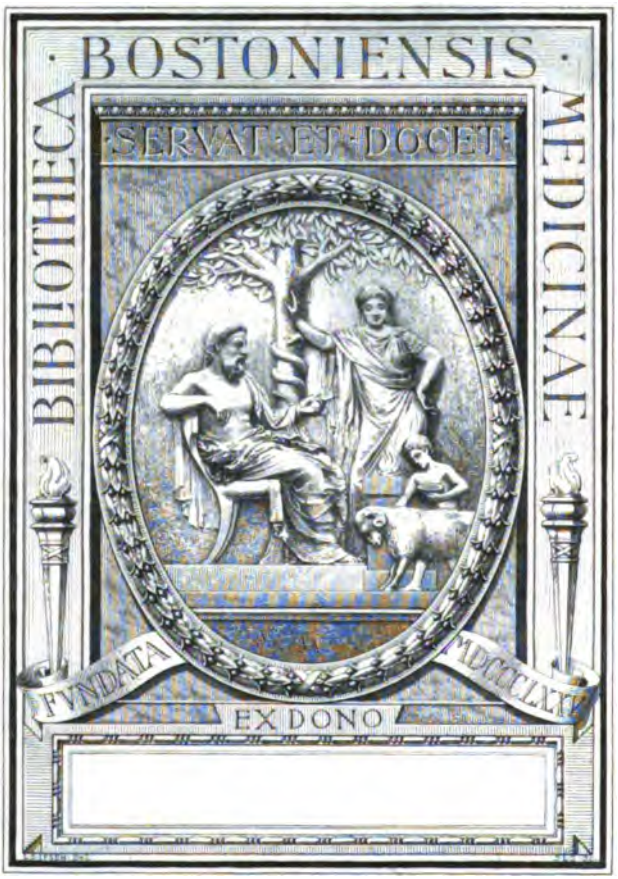

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EPILEPTICS

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DR. EVERETT FLOOD

PRESIDENT 1907

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TRANSACTIONS

OF THE

NATIONAL ASSOCIATION

FOR THE

STUDY OF EPILEPSY

AND THE

CARE AND TREATMENT OF EPILEPTICS

SEVENTH ANNUAL MEETING
RICHMOND, VA.
Oct. 24, 1907

VOL. V.

Edited by WM. P. SPRATLING, M. D.

Permanent Editor Transactions of the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics; Medical Superintendent of The Craig Colony for Epileptics; Member American Neurological Association, etc.

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PREFACE

TO VOLUME FIVE

The National Association for the Study of Epilepsy and the Care and Treatment of Epileptics was founded in May, 1900, for these purposes:

1. To promote the general welfare of sufferers from epilepsy.
2. To stimulate the study of the causes and the methods of cure of this disease.
3. To advocate the care of epileptics in institutions where they may
   (a) Receive a common school education;
   (b) Acquire trades;
   (c) Be treated by the best medical skill for their malady.
4. To assist the various States in America in making proper provision for epileptics.

Ohio, New York, Massachusetts, New Jersey, Texas, Pennsylvania, Indiana, Kansas, Michigan, Minnesota and Missouri have each founded State institutions of one kind or another for dependent epileptics. Alabama and North Carolina have now taken steps toward doing the same. Other states are about to follow. There are not less than 150,000 to 160,000 epileptics in the United States, less than 4,500 of whom are receiving proper State support, aid and treatment. Above all things the Association wishes to increase its *active membership list* that it may not only have the means for carrying on its work, but that it may also interest as many persons as possible in the philanthropic work it has undertaken. All who feel an interest in charity, or who are engaged in the scientific study of the disease, are eligible for membership.

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THE SEVENTH ANNUAL MEETING

OF THE

NATIONAL ASSOCIATION FOR THE STUDY OF EPILEPSY AND THE CARE AND TREATMENT OF EPILEPTICS

RICHMOND, VIRGINIA, OCTOBER 24, 1907

ADDRESS OF WELCOME

By Governor Swanson of Virginia

Mr. President, Ladies and Gentlemen:—

I have no address to deliver this morning. I find myself very much in the condition of the old negro preacher who divided his text into three parts. "First," he said, "I will talk a little of what I know a little about. Next I will talk a little of what you know a little about. And then I will talk a little of what we all know a little about." I shall have to confine myself to the last of the three.

I am glad of this opportunity of being present with you and meeting this assembly. We are glad to welcome you here.

There is no more important or generous work than the treatment of the insane and the epileptic, and as Governor of the State, I want you to visit our hospitals and asylums and criticize them, pointing out our weak points. We

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prefer your suggestions for betterment to your compliments, and we look for great benefits from your visit.

No body of people have done more for humanity than have the physicians of America, and they have done it with poor compensation. We all recall the story of the physician who sent his itemized bill to a patient—so much for visits, so much for medicines—and the patient enclosed a check for the medicines, saying "I will return your visits." Such is the pay the physician often gets.

When the Legislature meets in January I will use my best efforts in behalf of your work.

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ADDRESS OF WELCOME

By Mayor McCarthy of Richmond

Mr. President, Ladies and Gentlemen;—

There is a maxim of mathematics that the greater includes the less, and I am sure the remarks of Governor Swanson are sufficient to cover your welcome to Richmond. Still it is proper for one in my official capacity to represent the people of Richmond. We are glad to see you here. The Governor is the manager of the rural districts of Richmond, and while he welcomes you there, I welcome you to the heart of the Commonwealth. I am glad to know you are here for the comfort and relief, and possibly for the cure of those distressing persons, epileptics. I wish you could help some of us who suffer from eccentricities, etc., which subject us to severe criticism from newspaper editors. Those in official positions like the Governor and myself continually need the care of medical scientists. I hope the day is not far distant when the profession will realize that all require attention to the needs of the brain. Sufferers from mental disorders are, as a rule, the last persons to discover their affliction. I claim to be a constant sufferer, feeling my need of relief. I have read in the last week more about Epilepsy than I have ever read for fifty years before, and I have been an epileptic for the past week; when I read about tuberculosis, I always cough and choke.

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People who wish to be doctors have my consent. My association with them has not been pleasant and I have no fondness for their attentions, but at the same time I concur with the Governor when he said that doctors not only do more valuable and acceptable service than any other group of men, but are also the most unappreciated. Doctors receive, as a rule, neglect and ingratitude. I have often thought that the word "patient" should apply to the doctor rather than to the patient. I remember with great affection the doctor who served my father's family for fifty years, who gave his long life cheerfully to the service of the community, and who died without money to pay his funeral expenses. I wonder at the cold neglect of the world towards men of that sort. I remember after the war I had the pleasure of calling upon him and telling him that my brother and myself remembered his devoted attention to our father, and we had gotten together $50 or $75 in money and I handed it to him. The old doctor looked at me with affection and tenderness, and said; "My son, don't you think you will need this money more than I do?" I said "No, we do not need anything, clothes, house, nothing; we have no necessity; we are used to it; and we will esteem it a favor if you will take this money." He said "How about your sisters, don't you think they will want it?" I said "No, one of them is at a boarding school where they know nobody has any money and they don't expect us to pay, and the other is in the country where they don't need it;" so I said "Doctor, take this money and credit it on account." He said "If you must pay it, I will take it and count it square." Four years' service! He is sleeping now in Hollywood and I believe if Heaven rewards the faithful and devoted, he is enjoying the reward of the saints. I wish I were able to express

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my regard to those men who devote their lives to the relief of suffering humanity. I know of none who are so little appreciated, and whose reward is so little in proportion to service rendered.

I am here to extend to you a hearty welcome from the people of Richmond.

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ADDRESS OF WELCOME IN BEHALF OF THE MEDICAL SOCIETY OF VIRGINIA

AND OF THE

RICHMOND ACADEMY OF MEDICINE AND SURGERY

By Dr. Ennion G. Williams, President of the Richmond Academy of Medicine and Surgery

---

In the absence of Dr. Barringer, President of the Medical Society of Virginia, it becomes my pleasure to welcome you in behalf of the Medical Society of Virginia, and of the Richmond Academy of Medicine and Surgery.

Our medical organizations are for the purpose of developing scientific facts and for the promotion of their practical application.

The marvelous advance of civilization during the past fifty years is well known to be the outcome of the development of science and the practical applications of her teachings. All previous ages had their men of science, but these men were, for the most part, men remote from the practical affairs of everyday life, who, in the seclusion of their studies or laboratories, delved deeply into the scientific lore of their times, but regarded it as a degradation of their sacred work to seek, or even to allow for any practical application to the material pursuits going on about them.

But science today is on a far different basis. There are still a few scientists of this old school—scientists for the

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sake of science in the abstract—but now, no sooner is there announced a new discovery than the world asks, "What is it good for? What use can we make of it?" And at once thousands of men, in scores of fields, vie with each other in the attempt to put it into some practical application in their own lines of work.

That this profiting by the teachings of science has been the real factor in our recent civilization which has enabled it in half a century to outstrip, in material advances, all that had been done by the previous ages, cannot be successfully controverted.

In every industry this is true, and there is not one of them but owes its present stage of development to this fact. Everywhere this is the spirit of the age, but in one direction only does it lag behind, and this exception is found where we would expect to find scientific teachings seized upon and applied with greatest avidity.

The advances in medical science have been full as rapid and perhaps even more radical than those in any other field. The possibilities for good which have been brought within our grasp by the teachings of the new science of bacteriology alone, would, if fully taken advantage of, mean more for the prolonging of human life and the lessening of human suffering than the work of all previous centuries. Science has done her part, and right nobly has she done it, but those who alone have it in their power to put her teachings into application on a large scale for the benefit of the human race, have lagged behind. It is not here as with discoveries which are capable of industrial application; for every factory owner can at once seize these and put them into use—nay, he must do so or go under in the race for supremacy. But the teachings of preventive medicine can be put into effective operation by

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no small group of men who have power to act. They are for the nation, the state, the municipality, and with singular blindness our legislators have but grudgingly given scope to those who stand ready to do for the human race what every manufacturer can do for his business.

But it would not be true to say that nothing has been done to profit by the teachings of preventive medicine. Does not our national government spend hundreds of thousands for the eradication of hog cholera? Is there not, even now, at the river which flows by this city, a dead line over which no cow may pass during certain months of the year because of national quarantine against Texas fever? And is not this founded on the scientific discovery that this dreaded malady of cattle is due to a microscopic parasite conveyed by the bite of the tick?

Only when it comes to man, are our legislators slow to act. They can understand the economic status of the diseases of cattle: for cattle cost money, money is sacred, and therefore these diseases must be prevented, even at enormous temporary expenditure of the funds of the nation. But they will not see that the health of the community has an even greater commercial value. They regard man's diseases as a mere matter of sentiment, and with human heartaches they are not called upon to deal!

Year after year the effort has been made to give to an unfortunately afflicted class of our citizens in Virginia the benefits of humane treatment, but still they continue to be crowded into asylums for the insane, in surroundings the worst imaginable for the cure of their affliction, and so far little has been done.

Times are changing. A new era is dawning. We hear the good words of our Governor and Mayor. The time is rapidly coming when by the co-operation of legislators and

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medical scientists the preventable diseases will be prevented, the curable diseases cured, and the unfortunate victims of the incurable will have their pathways smoothed until they reach the place where there is no more suffering.

These being the common purposes of our organizations, we extend to you our cordial greeting and welcome.

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RESPONSE TO ADDRESS OF WELCOME

By Dr. Everett Flood, Palmer, Mass.

I am glad to have the privilege of responding, though briefly, to the courteous words of welcome spoken by the chief executive officers of your renowned state, your celebrated city and your learned societies.

The honor done this Association by the presence of such distinguished sympathizers is a feature to which we have not yet become accustomed, but it is a most encouraging indication of the growing esteem in which our work is held.

The credit for the inception of this movement is due to Dr. William P. Spratling of New York and such men as the Hon. William P. Letchworth, the first president of the Association, Dr. William N. Bullard and Dr. Henry R. Stedman of Massachusetts, Dr. Wharton Sinkler of Philadelphia, and other men of like prominence who were willing to associate themselves with him for the purpose of promoting the various interests outlined in the declaration of the new association.

Dr. Spratling, much to his own regret and more to ours, is unable to be present today. His written words will, however, voice his interest in this occasion and his appreciation of your hospitable reception.

From all these men named and from others, some of them not present, who deserve similar mention, and from every member of this Association, and from every person

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who shows his interest by being present today, as well as from the vast host of the afflicted whom we try to help, and from their multitudes of interested relatives and friends, we now voice to you our thanks for your interest and your help.

Proper justice cannot be done this portion of our theme if we fail to especially recognize in this present gathering the invaluable services of the chairman of our executive committee, Dr. William F. Drewry, who by patient and persistent efforts has faithfully served our interests and made the success of these meetings so assured.

To your local committee of arrangements and to such others as have co-operated with them, due recognition should be given most cordially.

Again, on behalf of our National Society, in my official capacity, in my personal appreciation, I return to your several bodies represented and to you as individuals, the gratitude and thanks of this Association.

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RESPONSE TO ADDRESS OF WELCOME

PREPARED BY W. P. SPRATLING, M. D.

Craig Colony for Epileptics, Sonyea, N. Y.

Governor Swanson, Members of the Association, Ladies and Gentlemen:—

I fear those charged with the responsibility of arranging the program for this meeting did not do the Association or its distinguished guests honor when they placed my name on the list of those whose very pleasant duty it is to respond to the hospitable addresses of welcome that we have just heard.

Never before in the history of the Association have we been honored by the presence of the distinguished chief magistrate of a sovereign State, by the presence of the executive head of a hospitable and beautiful city, and by the President of the highest medical body in the State in which we meet. Surely, we have come among friends whose generous and kindly welcome will inspire us to the utmost limit in the work we aim to do and whose good feelings, I can testify, the members of this Association reciprocate in the highest degree.

We have assembled in Richmond on this occasion to do all in our power to further the best interests of a class of the most sorely afflicted among all mankind—a class whose total number in the civilized world is known to exceed 3,000,000 souls, who exist in the United States to the ex-

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tent of more than 160,000, and who may be found throughout this and all other lands—in all communities, in every empire, state, county, village, township and hamlet—in the ratio of fully one to every five hundred of the population at large. So we have come to deliberate for a mighty host whose members are crying loudly for the best that charity, humanity and science can give them. These people are today knocking insistently at the doors of many states for the aid which every humane government should accord them without hesitation or regret. They stand today as they have stood for many generations past to appeal for relief from the fearful ravages of the oldest and assuredly the strangest disease in human history.

More than 2,300 years ago a man who led in ancient medieval thought and action wrote astonishingly well concerning the epileptic's strange malady and its treatment. At that remote period the very atmosphere about the epileptic was filled with mystery and with all the morbid fancies to which it seemed the tongue of mankind could possibly give utterance. And this strange mysticism, combined with all powerful superstitions and with an array of false knowledge rarely witnessed by man, controlled the treatment, so-called, of this extraordinary disease for hundreds of years.

Less than a long generation ago a kindly-hearted German Lutheran clergyman heard the stifled cry of the epileptic for aid and began at once to help them. He gathered together four dependent epileptics in a small cabin and ministered rightly to their needs. Shortly he built new houses, filled these with more people, instituted industries, enlarged his charity year by year and kept on growing, until today—forty years after its origin—Bethel Colony at Bielefeld in Northern Germany, the parent

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charity for epileptics, is known throughout the civilized world.

Next the movement crossed the Atlantic Ocean, and Ohio founded a hospital for epileptics at Gallipolis—an institution which today has more than 1,300 patients and which long ago took its stand among the best of its class to be found in the New World.

Next came New York State with The Craig Colony for Epileptics at Sonyea, which remains the only true Colony for epileptics in this country to this time, for it consists of an aggregation of small homes, with complete liberty for all who can appreciate it and properly use it as the keynote of its organization and of its existence. Nearly 1,100 patients are now under care and treatment at Sonyea, and work is going forward as rapidly as possible for the ultimate care of probably as many more.

Massachusetts was next to fall in line, with the result that at Palmer there is a splendid institution, growing year by year, for the treatment of this afflicted class. Over this institution our esteemed President has the honor to preside.

Next, New Jersey founded at Skillman, near Princeton, a Village for Epileptics which is rapidly approaching the ideal for an institution of this class. Then came Texas, also, with an epileptic Village at Abilene.

Next, Pennsylvania with an institution at Oakbourne, mostly sustained by private aid through the munificence of a single citizen of Philadelphia—Mr. Henry C. Lea.

Then came Kansas, which founded at Parsons the first institution for epileptics in the middle west.

Two years ago Indiana purchased a tract of more than 1,200 acres of land on which to build an institution for epileptics, and the Governor of that State recently issued a

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proclamation declaring the institution opened for the admission of patients.

Pennsylvania has taken another step forward and founded an institution devoted to the combined care of feeble-minded and epileptic, and it is hoped by some of the friends of the new institution as well as by the members of this Association that it will not be long before the institution at Spring City will be given over wholly to epileptics; for students of this disease think that the epileptic cannot satisfactorily be cared for in an institution in which there are other defective classes cared for at the same time.

SCIENTIFIC PROGRESS

In addition to material progress so far as it relates to the public care of the epileptic and his education, there has been a positive advance in the study of this disease along scientific lines.

Eighteen years ago there was not a physician or scientific man in the United States whose entire time and every effort was given up to the study of this disease. Today there are not less than twenty-two physicians whose work embraces absolutely no other line of effort. At Sonyea a laboratory is maintained by the State under the supervision of a competent pathologist solely for inquiring into the causes of this disease. At the same institution more than 150 epileptic children are in manual and ordinary schools, while good schools have also been started at other places—especially at Palmer, Mass., and at Parsons, Kans.

At Sonyea a systematic attempt has been going on for years to ascertain the true value of surgery in the treatment of epilepsy. Last year 172 capital surgical operations were performed by members of the medical

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staff. Valuable papers have been read by leading surgeons before this Association during the last three annual meetings on the same topic, and no one can doubt but what these studies and discussions will bring valuable results in time.

The question of the value of pathological anatomy and histology in studying the causes of epilepsy is a serious one. Personally I have long felt that science will never profit to any considerable extent in its effort to locate the true causes of epilepsy by studying dead-house material alone. It has been my conviction—and that conviction has been growing for years—that most of our studies to ascertain the cause or causes of this disease must be made upon the living individual. That is the trend of laboratory work at Sonyea today.

The curability of epilepsy is no longer questioned; it is now only a question as to what per cent can be cured. But that is a question we cannot answer yet, for independent workers who write down their observations must learn to crystalize and make uniform their reports. We must report like cases alike, and so rid our statistics of the inequalities that make them almost useless for purposes of comparison. But it can be written today that 7% to 10% of all epileptics may be cured if they will come under treatment of the right kind long enough, and stay under it as long as conditions demand. Many who cannot be radically cured may have their attacks arrested for life or so long as the individual will live in the right way. Freedom from convulsions is more often a matter of right living than anything else.

All this sounds like progress and such it is; good, steady, and I believe true and along right lines. The picture of the accomplishments of the past eighteen or twenty years is gratifying indeed;—but what of the

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future? 160,000 epileptics in the United States and less than 3,500 under proper care; forty-six States in the Union, and yet institutions for epileptics maintained in only seven of them. There you have an indication of the work of the future. We must begin anew. We must do more. We must do our part. We must point the way and then do all we can to have others follow. Annual meetings like this do great good—especially when local feelings are such that they create a pleasant feeling—even a feeling of enthusiasm—and cause us to think that here is a community whose sympathy is with us—here is a community whose public leaders in our work strongly wish to fall in line with what has been done by the best States in this direction. Dr. Drewry's work in Virginia will not be forgotten. Here is a helping hand of the strongest kind. I bid you let us grasp it because it helps to lead us to a higher conception of duty and to work better done. Let us help them to the same plane. Co-operation is the word. The ancient saying that in union there is strength is truer today than ever before.

In conclusion, we thank the people of Virginia, especially its high executive officers and its well-known scientific men, for the welcome they have given us today, for the stimulus and sympathy of their presence. You do not know what you have brought us. Maybe you do not understand how a word of encouragement helps. And now turning our faces to the horizon of our work, we call to your attention the fact that we are banded together for these purposes, and again we ask your aid:

1. To promote the general welfare of sufferers from epilepsy.

2. To stimulate the study of the causes and the methods of cure of this disease.

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3. To advocate the care of epileptics in institutions where they may

(a) Receive a common school education;

(b) Acquire trades;

(c) Be treated by the best medical skill for their malady.

4. To assist the various States in America in making proper provision for epileptics.

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PRESIDENT'S ADDRESS

By Dr. Everett Flood, Palmer, Mass.

To approach this subject under any circumstances is a fearful thing, but to attempt in this presence to say an authoritative word is appalling.

It is quite natural that the medical fraternity should look to centres for special cases, for information as to the progress made by medical science in the care of their own kind of cases.

We have in this country certain men who can give us information as to the subject of aeronautics. It is a difficult theme and we are not much disposed to grumble if we do not get very detailed instruction as to how to navigate the air.

We do not cure all cases of pneumonia, typhoid and tuberculosis, adjust perfectly all breaks in the skeleton, treat to perfection the serious results of accidents, obliterate the scar of extensive burns or do all to the extent of desires for hundreds of serious diseases. Why is it a discredit to us that we do not cure all cases of epilepsy?

In our study of any condition, the best we hope for is to know what has been done. Facts must stand for what they are worth. We can hope for new facts from time to time and point with expectation to the probabilities. How to treat idiosyncrasies and various sorts of family

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peculiarities, the general subject of inheritance, effects of mixture of blood, miscegenation and a few thousand other undeveloped lines of research would have its interested followers.

If not very much is developed as to these matters, we do not criticize unfavorably, but lend a hand to see if other facts observed by us may not lead to new and more useful deductions. All our work for the epileptic and all our efforts with the insane yield us only unsatisfactory results. We must not, however, cease our efforts. If under all such conditions it is impossible to come out often with wonderful discoveries, we should not be denied the credit of doing a lot of plodding work from which in time some results may be shown.

I think some of this apologetic foreword may apply to those who are working in the special line of the care of epileptic persons. We have to count the possible disadvantages and the obstacles to a beginning movement which must always be met.

First, it is a matter of moment to get the public interested so that buildings will be granted. Then a study as to the kind of buildings. The accommodation of the numbers who are eligible. The matter of hiring suitable helpers at the price within our means, the sorting out process so that we may finally have cases not too far advanced to preclude all possibility of study, the organization of a great family with ten thousand intricate interests and cross purposes.

The position of this Association among medical societies is not altogether clear to many. We are an Association small in number, we must comprise practical business administrators, expert neurologists, acute psychiatrists, and philanthropists rich in goods or in good purposes, or

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both. The fact that our medical work must of necessity be carried on in a line with our sociological makes it necessary for our transactions to cover a much broader ground than would be the case if we only treated our cases medically.

A great deal has already been accomplished by the Association. To enumerate how much would be to recount the well known advances of the last few years in this and its allied lines of research; to tell of the new construction for care of epileptics, the plans for the future and the efforts of individuals now working to bring about the ideal in their own State.

What it ought to do further is well worth our consideration.

The purpose of the Association is well enunciated in our program. No better statement can be made of what we hope to do.

Objects of the Association:—

1. To promote the general welfare of sufferers from epilepsy.

2. To stimulate the study of the causes and the methods of cure of this disease.

3. To advocate the care of epileptics in institutions where they may

(a) Receive a common school education;

(b) Acquire trades;

(c) Be treated by the best medical skill for their malady.

4. To assist the various States in America in making proper provision for epileptics.

I wish I were wise enough to enumerate all the lines of investigation for our Association. This I would leave to others. I can only suggest a few points which have impressed me.

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It should be the function of a National Association to take a broad and unprejudiced view of the situation, and it will be difficult to add to this general declaration of principles. For immediate detail I believe this Association ought to define epilepsy. The difficulties are many, but I believe our neurologists should stand forth boldly and say that a fit under any conditions is always brought about by the same mechanical action. The cause may be stated as tetanic, eclampsic, gross pressure on the brain, inheritance or what not.

We should know whether the convulsion is a symptom among other symptoms of a variety of conditions, just as headache is a concomitant of a variety of things, or a condition so far by itself as to merit a distinct nosology. No accurate statistics can be made unless we all work from the same classification of data.

I believe when some of the elementary principles can be settled that we can simplify our study much, even if we do not alter the terms in which we express ideas.

Epilepsy may still be epilepsy but with an extended and definite application.

The Association ought to define the legal status of the epileptic. It ought to demonstrate beyond a doubt that any person who has habitually suffered from fits and the states of mind accompanying them, especially if the person has become a public charge because of his inability to keep a position and earn a living, is irresponsible and insane.

The importance of fully nourishing the epileptic, or better, of properly sustaining the growth of the child liable to a breakdown, cannot be overrated. This means a suitable parent, one who is able to fight not merely intemperance, but who believes alcohol to be a poison and unfit for use as a beverage; one who knows enough to keep clear of

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wrong and to teach how to prevent all venereal disease by the prophylaxis of early marriage and by moral development, as well as by all other means.

Such a parent would forestall tuberculosis by both these means, and his children would build solid health where now prevail multitudes of disorders of which convulsive attacks are symptomatic. How unaccountable that the human body is so abused by the one who is most interested in it. We cannot for an instant believe that such recklessness is exhibited except where ignorance of results exist. We can imagine a state of life where everyone enjoys perfect health and where each one is contented with the enjoyments of such a state.

Dr. William Hanna Thompson in an article in "Everybody's" July, 1907: "The powerful centres in the spinal cord would rack us to pieces but for their being checked by the brain above, and they actually do so when the poisons of strychnia or of tetanus overcome the restraint of the brain."

In the predisposition to epilepsy, some pitch over the brink easily, some resist strenuously. Some eyes accommodate better than others and are almost never taxed beyond their capacity. In a like manner, some people endure more than others. There are those who appear never to endure. They remove themselves from troublesome environment. They do change places often; but the long suffering may well envy their evasive methods.

To recur to the former thought I repeat that the prevention of epilepsy touches sociology; it must go hand in hand with it. We must consider how parents should rear their children. We must so arrange that they can give a great amount of time to them. To teach them right they must themselves know right.

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All this involves: 1st. Goodness on the part of the parent. 2nd. Leisure for attention to children. 3d. Sufficient support by reasonable hours of labor. No man can rear his child properly if he must work over-long hours.

With all these difficulties before us the wonder is that we have courage to go on. But this may as well be said of sociological problems in general, so that we have to consider in what a hard field we work and take some courage from the evident fact that by keeping on we do make progress.

Most cases of beginning epilepsy are traceable to an early meningitis, probably with the predisposition of inheritance. So long as the care of children is so inadequate we can not hope for a stay of the trouble. How many cases are preventable by intelligent care, how many ignorantly lighted cases of cholera infantum lead to an infectious meningitis, we can hardly say; but we know that the parent is often the cause through ignorance of the ruin of his child's health and mind. We must labor to allow the parent who is sober and willing to have suitable time to rear his children. The gradual evolution through millions of years works out where advance has already made the universal plan of intelligence beyond our comprehension. With such breeding as we have hinted above we must expect every kind of vagary.

I would like to be able to say to you that epilepsy is not a disease; that it is always a symptom of an underlying condition; that you may probably find that underlying condition and name it and treat it; that the fit is a symptom of the nervous disease. I would like to be able to say that evolution will explain all the anomalies of the epileptic condition. Perhaps the physical and the mental

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are so nicely balanced in all highly organized creatures that a slight disturbance will throw it out and the higher centres are unable to control the energy of the powerful lower centres. We observe it in all highly organized animals. Even in vegetables we may demonstrate, as is shown in plant response, a nerve element giving much the same response in kind from stimulation, as we get in animal tissue. Mineral substances are not altogether exempt. A unity of nature may be demonstrated and a great advance may yet be made in explaining causes, if not in advancing treatment. Meanwhile, we must ask the public to be patient. Our men are not idle, and in time, a tedious time though it may be, results of importance will come forth for the further amelioration of the diseased condition of mankind.

Principles of evolution have now become established to such an extent that we may make the claim of its causative relation to all mental and nervous diseases, if not to all of a more positively physical order. The principles involved have been in existence forever. They have worked slowly just as they are now working. Whether the end has been reached in some instances is not for us to decide. Thousands of millions of years may be involved, but nature seems to prepare for a change a long time and the actual visible change comes before our attention swiftly. We all at once realize the change.

Time reveals the instability of the highly evolved life. Great possibilities for development open when perfect health can be maintained, and great dangers open in the retrograde changes coming in the revolutionary processes. The by-product of evolution must eventually become extinct. Meantime, our descendants may meet, in isolated cases, worse specimens, more specialized deviltry than we

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have yet seen. Who can say that the potential angelic being is not in existence, the summit of his glory to be within the range of possibilities millions of years hence. Certainly we can fear the diabolism of the offspring of some varieties of humanity already extant. The improvement of our race must come from the gradual ascendancy of this evolving angel; the undesirable element slowly fading out, with here and there a burst of astonishing but erratic genius. From this point of view, four out of five of the persons we meet are absolute monstrosities in mind or feelings or will, as well as in body, compared to what they might be and ought to be. Inheritance and environment have interfered with the normal advance, while ground is being lost in hundreds of the lines which are doomed to final extinction. Glorious ascendancy is coming to a few lines of development.

In order to have knowledge of epileptics, several kinds of experiences are needed; one must see epileptics individually, treat them in attacks, watch over them in status, associate with them when at their best, see them in masses under all their conditions of life, manage their general modes of life, arrange their work and their play, see them in anger, know what motives act upon them and be their friend. There are many matters relating to epileptics which investigation has determined. We may well feel pride in the patient work which has been done and at the possible results to which this work leads.

A matter not fully settled by accurate statistics, but well within my belief, is that fifty per cent of persons who have fits recover. A large number of spontaneous cures occur, so large a number that it is a matter of little moment to the average mother among the poor if a convulsion occurs. They expect the child to outgrow it. This almost universal

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belief has reason for its existence. There are large numbers of unreported cases. We get evidence of this every day. Parents do not recognize the condition or they conceal it, so that this element has to be considered as an important one in collecting statistics.

In the care of epileptics, I ask, what do we desire? The answer should be, nothing further than the welfare of our patients. We would cure every case, prevent every other one, or in other words, we believe in carrying on two lines of warfare. First, to better the condition of each sick one. Second, to prevent others from becoming sick. Our effort is then to discover what is the best thing we can do for our patients.

In this matter I call attention to the unselfishness of men who work in this line, though we cannot claim any superiority in this over other classes of conscientious workers. If we find a useful remedy we give out the formula. The commercial instinct is dead within us, and it is to our credit that none but medical men would do so.

Finally, I believe in the human race. Work with defectives is not discouraging but to the optimistic observer it is full of hope. Of course, anybody may lie down and die of despondency; but we are not of that hue, and this Association has a work to do to keep the color right and to recruit our ranks from all classes of society.

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THE VALUE OF ELIMINATION IN THE TREATMENT OF CERTAIN FORMS OF EPILEPSY

By J. Allison Hodges, M. D., Richmond, Va., Professor Nervous and Mental Diseases, University College of Medicine, and Physician-in-Charge of Hygeia Hospital

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If the etiology and pathology of Epilepsy could be definitely determined, its treatment could be logically defined. The former, however, being uncertain with our present knowledge, the lines of treatment pursued are necessarily varied, and to some extent, empiric. Consequently, in the absence of a well established and fully verified hypothesis as to the cause of all the different varieties of epilepsy, every practitioner must be guided in the treatment of the special forms of this disease largely by his personal experiences.

In the study of the large majority of cases which have come under my personal observation, I have been impressed with the toxic pathogenesis of epilepsy, and my experience is highly corroborative of the old conception of the disease, in which faulty chemical processes were believed to play an important and conspicuous role, for in nearly all, there has been a hypertoxic underlying basis with inadequate elimination.

I am, of course, perfectly aware that this theory does not embrace entirely the causative factors in all cases of epilepsy, but am persuaded that it does in most of them, and even more frequently than we generally believe.

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When the primal etiological factor shall be definitely determined, I believe that the most tenable theory as to etiology will be a condition, I shall not venture to say just what, that will be at least dependent upon deficient elimination, with resultant toxic effects upon the nerve centers. If this be true, then elimination is of the utmost importance.

As a general rule, we are content to employ only such remedies and measures as will afford temporary relief and ameliorate the paroxysms, and we do not, in my opinion, in general practice, give that careful and systematic thought to the avoidance of recurrences that we should. I do not offer you any new thought along this line—the facts are well known to you all—but I would insist that we often practice least what we know best. It is also true that every one of us practices this general method in a general manner, but we too seldom utilize it in a special way upon a special patient. I do not mean to be captious nor critical, but pointed and practical.

Believing that there are many estimable and scientific methods of treatment, I do not propose to be dogmatic, but would insist that, from personal observation and experience, the value of elimination, continuous and prolonged, in the treatment of this disease has not been sufficiently insisted upon by the average physician.

We all admit in a general way that the regulation of the primae viae is one of the essential basal principles of all well ordered therapeutics, but in the treatment of epilepsy do we fully appreciate its real value and efficiency? I am constrained to say that not until recent years have I appreciated this fact and had its full therapeutic value firmly forced upon me.

The old aphorism: "Clean out, clean up, and keep

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clean,'' has been relegated to surgical systems for too long. This maxim is of equal, if not greater importance to the physician, and especially to him who treats epileptics. This lesson was first taught me when I began regularly to treat my epileptic patients along special hygienic and dietetic lines; it was later more fully learned when I found that the less cause I gave a patient for auto-intoxication, the less frequent were the nerve explosions, and finally it was indelibly impressed upon my convictions when, with improved methods of elimination, even more satisfactory results were obtainable.

I would not have you believe that I am such an enthusiast as to believe that this one method is a cure-all, for it is not; but of all methods and measures known to me, it is the best, if conscientiously administered and faithfully and continually carried out.

Believing that there was more of utility and value in it than could usually be obtained in intermittent office consultations and prescriptions, and wishing to test this method to the fullest extent, I chose last October from my practice, three children, two boys and one girl, all of nearly the same age, twelve to fourteen years, and sent them to my private hospital, where I usually do not take such patients at all, for a term of six weeks.

So far as I could judge from office consultations, irregular and otherwise unsatisfactory, they were nearly on a par as to the common expressions of the disease, such as the nature, frequency, etc., of the attacks, their average paroxysms being from two to four a week, when not under large doses of the bromides. The preliminary urine examinations showed in each case an undue proportion of indican in the renal secretion, as well as a deficient secretion of the urea.

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It is needless to trace the treatment of these cases minutely—it would be as tedious to me as it would be tiresome to you; but with the exception of small doses of strontium bromide and a gradual reduction of salt in an aseptic diet until complete dechlorization was secured, these patients had no medicines given except a laxative when necessary for the elimination of the various products of metabolism.

All of the other methods available, however, for the purposes of elimination were employed, such as an aseptic diet, free water-drinking, moderate exercise by means of walking, massage and vibration, sweating by means of the Baruch tonic bath system, and eliminative measures by aid of the sinusoidal, high frequency currents, etc.

The result was that in each case the seizures were notably decreased and the general health markedly improved. The vital and resistive force in each patient was so much improved as to be readily noticed by any one, and in the case of one of the patients there has been up to this time no return of the epileptic seizures, while the other two have suffered only slight inconvenience at long and irregular intervals.

None of them have exceeded fifteen grains of strontium bromide daily, and all have continued, as best they could, the eliminative and tonic measures first prescribed. These cases cannot yet be classified as "cured," but their continued improvement after the lapse of a year argues well for the future.

It is eminently true of these cases that they were all of undoubted toxic origin, but be this as it may, it may nevertheless prove that there are many such who, under similar ideal conditions, which are simple and accessible to all, if we only consistently carried them out, might

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likewise get as beneficial results. That the restrictions of the regime under which they temporarily lived was of benefit, I do not for a moment deny, but I also claim that if we, as physicians, exercised that same "eternal vigilance" of these cases, as, for example, we do of our typhoid patients, that the conditions named above are not so difficult of employment or realization in our daily practice, and that our epileptics would be largely the gainers thereby.

Neither do I mean to prove by this inadequate test that "three soldiers make an army," and that all such patients can thus be pronouncedly benefited, still I do believe that a feather may show which way the wind blows, and as I said in the beginning, this discussion is intended to be not dogmatic, but suggestive; not critical, but helpful.

The inefficiency of treatment in the past should, I believe, at least lend credulity to the present and hope for the future.

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THE SYSTEMATIC TREATMENT OF EPILEPSY VERSUS ITS TREATMENT BY AN OCCASIONAL CONSULTATION AND PRESCRIPTION

By William P. Spratling, M. D., Medical Superintendent The Craig Colony for Epileptics, Sonyea, New York; Member American Neurological Society; Member New York Academy of Medicine; Permanent Editor Transactions of the National Association for the Study of Epilepsy, etc.

A more hopeful tone is steadily becoming apparent in the opinions and writings of neurologists whose work in the field of medicine leads them into daily contact with epileptics of any considerable number.

We constantly hear less discouraging statements relative to the supposed incurability of epilepsy since the epileptic became a public charge in several States in this country ten to fifteen years ago, and, as such, found themselves in a position where they could be constantly under skilled observation and subjected to more systematic and continuous treatment than was possible when the patient was not under a watchful system of semi-restraint.

The creation of special institutions has done more to prove the curability of epilepsy during the past quarter of a century than was accomplished by any system or method in vogue prior to that time.

Although many of the older writers on epilepsy whose work dates back some seventy-five or one hundred years asserted epilepsy to be curable in 30% to 50% of all cases, it is regretable that no authentic detailed records are to be

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found in support of such assertions. Medicine has become too exact a science to permit our accepting statements that are not supported by conclusive clinical data.

Beginning with Ohio in 1890, then New York, Massachusetts, New Jersey, Texas and Kansas, a line of State institutions has sprung into existence in which there are at present in the vicinity of 3,500 patients. The medical officers of these institutions act in concert in asserting that the prime feature essential to the treatment of epilepsy is the control of the patient. They are unanimous on this point. And while these physicians have had the power to a certain extent of holding patients under treatment a sufficient length of time and subjecting them to certain forms of treatment which would have been impossible were the patient in his own home and to a large extent master of his own actions, they have had opportunity to compare the results of their work under these conditions with the results of work performed by them at a prior time. It is the ability to compare results acquired in the two ways that makes their later opinion valuable.

We have only to consider the causes of epilepsy to understand the necessity for the complete control of the patient during treatment. No well-informed physician—certainly no neurologist or epileptologist of this age—will deny that heredity plays a more important part in the etiology of epilepsy in one way or another than is played by any other single factor. It has been ascertained at The Craig Colony that 16% of all epileptics out of a total of 2,300 admitted since the Colony was founded, suffered from epilepsy because they had either an epileptic mother or an epileptic father.

The very fact that epilepsy was the leading cause in the production of their epilepsy is prima facie evidence that

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the disease in question is not one to be satisfactorily treated with an occasional visit to the physician's office. It is apparent that much more must be done, and *that* much more lies first in being able to absolutely control everything the patient does. We have also come to know that the life habits of certain individuals play a leading role in the production of diseases, and these habits show their importance in no disease more strongly than in the diseases of the nervous system.

Epileptologists recognize the final results of "food-poisoning" as a pregnant cause of epilepsy in many adults—especially middle-aged men. This cause is ably seconded and increased by alcohol in a slightly smaller percentage and frequently in the same individual.

No matter how explicit are the directions we may give a patient living in his own home in regard to food and drink, he is not bound to carry them out, and unless he be a person of superior intelligence and possessing a genuine desire to recover from his disease, he is too prone to permit his pernicious appetites to govern his future condition. I have witnessed the truth of this assertion in scores of cases. I have come to understand that a very large percentage of patients who have been under care and treatment at The Craig Colony can live under the semi-restrictive influences of Colony life month in and month out—sometimes year in and year out—absolutely free in every respect from epileptic manifestations of every kind and degree; but the moment such a patient is permitted to go home, the moment the withholding influences of Colony life, which are all for the good, are removed, that moment the attacks are invited to return and this they generally do. Such facts as these constitute the clearest argument within my knowledge it is possible to adduce as to why

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epileptics cannot be satisfactorily treated in their own homes. This assertion cannot cover every case, but I have no hesitancy in declaring that it applies without exception to the vast majority.

Another noteworthy point is this: The best epileptic statistics available show that close to 85% of all persons who suffer from epilepsy acquire the disease before the twentieth year; and since the power to reason and to exercise good judgment and discretion in matters of personal hygiene are not given to persons in their minority, save in very rare cases, we have a second cogent argument for desiring to protect epileptics from themselves in cases where the lack of salutory supervision would mean incalculable injury to the patient.

Few epileptics who become such in childhood reach adult life in full possession of reasonably sound powers of mind. If the mind is not congenitally deficient—as it so often is in epilepsy—the effects of the disease make it so sooner or later to some extent in substantially every case; and since feeble-mindedness is a reason "a priori" for effective supervision, we have a third reason for recommending it as a necessity in the treatment of epilepsy.

The only way in which the systematic treatment of epilepsy can be secured is to remove the patient from his home, or to place a new factor in the home in the way of a competent attendant, a nurse, or a physician, either of whom should assume complete control of everything that pertains to the patient's welfare. Few instances arise in which the constant employment of a physician in the home for any length of time is possible. The cost of such a measure is generally prohibitive. More often an efficient, tactful nurse, trained in epilepsy can be employed; and when the patient's means are still more

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inadequate, an attendant or companion must then suffice.

The object is to treat the patient independent of parental sympathy, which, however essential and pleasant it may be for the patient, too often defeats the attainment of medical and disciplinary aims. One not bound or influenced by kinship ties to a patient is in a better position to apply mild measures of discipline than one who is controlled by such ties. I have rarely seen a parent who could deny the request of a sick child for some forbidden article of food, justifying it by saying "A little will do no harm this time." And this attitude pertains in many other ways. It is so common, so universal in fact, as to make the systematic treatment of epilepsy in the patient's home well-nigh impossible.

A final argument against home treatment is that epilepsy is a peculiarly *dangerous disease*. Anyone who has it may die at any moment as the result of a single seizure. In the records of more than 300 deaths in epilepsy it is found that more than 4% occurred as the result of a single seizure. A very much larger per cent die from status epilepticus, which is the bane of every epileptic's existence. This continuous epileptic state demands instant and vigorous treatment by a skilled physician to save the patient's life. It calls for the use of agencies not usually found in private homes but which every well-regulated institution public, or private, should provide.

The foregoing constitute the reasons in part why it is not feasible to properly treat epileptics in their own homes, and why the disease cannot be successfully combatted by having the patient make an occasional visit to the physician's office to receive general instructions as to diet and a prescription intended to overcome far-reaching

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conditions that cannot be removed except under two or three years close attention, treatment and observation while the patient is under effective control.

Like the treatment of tuberculosis the treatment of epilepsy must be diversified and far-reaching, and nothing short of a system in its treatment will secure the best results. Under a proper system 8% to 10% of all cases can be cured.

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ALCOHOLISM AS A PREDISPOSING AND EXCITING CAUSE OF EPILEPSY

Dr. Thomas C. Fitzsimmons, Wilkesbarre, Pa.

Mr. President, Members of the Convention, Visiting Ladies and Gentlemen:

I trust I may be excused for threshing over again such an often beaten subject as the relation of Alcoholism to Epilepsy; but the topic is such an important one, the relation so intimate and serious, ramifying in so many directions, that I may be pardoned for adding a number of examples from my own experience in confirmation of what has been said by so many observers before me, including Martin, Nothnagel, Echeverria, Wartman and others, as well as the many eminent men of this Association, for although alcohol as a predisposing cause of epilepsy has been recognized again and again by men who have had extensive opportunity for its intimate study, yet it cannot be repeated too often, and the lessons drawn therefrom cannot be inculcated too much.

This, to me, is one of the great, and grave facts that ought to be known.

Alcoholism as an exciting and predisposing cause of epilepsy, then, is not only a double danger—a two-edged sword cutting right and left,—not only causing epilepsy in the drunkard himself, but is also the reason for its transmission to his progeny; but as if this was not enough, an

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eminent Philadelphia physician in a paper read before the American Medical Association some years ago, said: "Man or woman does not need to be either epileptics or chronic-drinkers in order to transmit this distemper to his or her progeny, but that otherwise temperate people, if under the influence of alcohol during conception are then in a state calculated to produce epilepsy in children conceived under such conditions."

This aspect of acloholic toxemia in its connection with epilepsy as far as I know had not been referred to by any other writer. Yet I have also noticed it in more than one instance (in my early practice, as the more intimate life of the village enabled me to see things often concealed in populous towns). Recognition of the significance of this observation is of great importance to us as custodians of the public health, and cannot be emphasized too much.

Before presenting my own cases, allow me to draw your attention for a while to what has been said in the same line by others. In a series of 572 cases of epilepsy studied by Echeverria he discovered that 259 of them were due entirely to alcohol; 126 to alcohol and syphilis combined; forty-two of the series were credited to traumatism, and in the remaining 145, sun stroke, ague, excessive use of tobacco and mental anxiety were assigned as the apparent causes. Thus according to this observer nearly fifty per cent of all epilepsies were due directly to alcohol, and about seventy-five per cent to alcohol and syphilis together. Besides, in the families belonging to the first class—those where alcohol was the only cause of the affliction—thirty-nine per cent of the victims were also congenital idiots. In the second class—those where alcoholism was combined with syphilis—twenty per cent of the fathers and thirty

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per cent of the mothers were epileptics as well as inebriates.

Alcoholic epilepsy, as is well known, is frequently associated, too, with delirium, hallucinations and dementia, which would thus show a close relation between delirium tremens and epilepsy. In writing of the comparative danger of various sorts of drink, Echeverria states that he found that whiskey drinkers were among his worst cases. My experiences convince me that it is not in the sort or variety of alcoholic taken, but in the proportion of alcohol ingested, for German writers have found the constant drinking of beer a fruitful elicitor of epilepsy. Frenchmen claim that beverages flavored with aromatics—alcohol with aromatic trimmings—viz: absinthe, was most powerful in the production of epilepsy. Russians from the vineyard districts give the palm to freshly fermented wine. In all cases it is just the alcohol, and the variety of vehicle it is taken in does not matter.

Dr. Moeli, who has made researches in this direction in Germany, says that inherited epilepsy from alcoholism is common in the Fatherland, and that besides the commonly recognized epilepsy, from thirty to forty per cent of all persons with delirium tremens are also epileptics. In France, Hippolyte Martin analyzed 150 insane epileptics at the Salpetriers and found that sixty-six per cent were *positively* due to intemperance in parents and that in the remaining thirty-three per cent alcoholism as a cause was *only* not so clearly defined.

By way of additional emphasis to the subject of alcoholism as a predisposing and exciting cause of epilepsy, I have selected from my own practice a few cases from the many of which I have kept record and in some of which I have traced the hoof-prints of drunkenness as the parent of epilepsy, through three generations.

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The first epileptic coming under my lay as well as professional observation, because I knew him when I was a boy, was W., who was employed as a laborer upon my father's farm. I vividly recall the convulsive attacks that seized him on frequent occasions. This man was addicted to the excessive use of alcohol in the form of whiskey, and always after a debauch he had attacks so much more frequent as to attract notice to the fact that their increase in number always followed prolonged intoxication. He had an inheritance of alcohol that we could trace back for three generations. He came from a line of ancestors that were even remotely given to intemperance; besides, one daughter was the victim of an epilepsy that resisted every remedy. Finally she got married, when the seizures subsided for a while, only to return with increased force. When last heard from, although the mother of seven children, she was still an epileptic. As his relatives lived in our neighborhood I could easily trace back his history. His maternal grandfather was addicted to the immoderate use of alcohol, and his great grandfather was a common drunkard. One son became a drunkard without epilepsy; the daughter developed epilepsy without becoming a drunkard.

George—was the son of wealthy and refined parents, yet from his eighteenth to his forty-fifth year he was so much the slave of drink that he seldom went a week without intoxication. In his twenty-second year he developed spasms, having from then until his death an average of two weekly. He was of New England ancestry, tracing his lineage back to Plymouth Rock, and from a stock in which alcoholic addiction is not frequent; yet with the exception of his father and mother the ancestors all drank immoderately, transmitting to many of their progeny that weakened state of the nervous system which under the toxemia of alcohol broke out into convulsions.

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A great-grand-aunt, a grand-aunt and a daughter were epileptics.

Joseph J. Aet. 27. When he arrived in this country from England supposed to be temperate, he became a track hand on the Delaware and Hudson Railroad, but was in reality a secret excessive drinker. During the time he was under my care he had an average of five seizures monthly. Medicine had no effect on them. It is difficult sometimes to get the whole truth from the family about epileptics, but from this man's compatriots I learned that his father and mother were temperate but neurotics, his grandfather was a periodic drinker, during which periods he had "weak spells," evidently psychic epilepsy, and it was always after these periods of excessive drinking that they occurred with most frequency.

Robert M., son of a hotel keeper, was one of a family of eight. His father, a man of intelligence who nevertheless appeared to better advantage partially intoxicated than sober, as persons of certain temperaments sometimes do, his mother and sisters superior people physically and mentally healthy. The mother informed me that until her pregnancy with the boy she exercised the greatest marital precaution, not permitting his approaches until the father had gone two weeks without drink. Before the conception of her son this precaution could not be taken; was impossible because her husband had been on a protracted debauch and became uncontrollable. Robert was the consequence. In his twelfth year, without an apparent cause, he became epileptic. He was found dead seven years afterward in a workshop where he had fallen in a convulsion. Traced back, I found that his great grandparents on his father's side were excessive drinkers, but aside from this case there was no epilepsy in the family. Robert's sisters were models of physical and mental health, excepting one sister who after her marriage and motherhood became

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insane and was placed in an asylum, where she died recently.

John B. was the fourth son of sober parents. At the age of sixteen he developed his first attack of epilepsy. Next day his father, an intelligent laboring man, brought him to my office and gave me all the information bearing on his ancestry that he knew. His father was a total abstainer, but his grandfather was a confirmed drunkard. There were no other mental defects discoverable in the family. John B. responded poorly to treatment, the convulsions increased in numbers and severity until I lost sight of him four years ago, at which time he was a confirmed epileptic.

I shall now cite two cases—boy and girl cousins—of extreme interest in this connection, children of a sister and brother respectively. They are about the same age and the same description may answer both. John W. is now a boy of fourteen years. At the age of four he fell from a distance of about five feet, striking his head and shoulders. I saw him soon after the fall, but discovered nothing unusual in the case. No marks where he had struck, no evidence of either internal or external injury. About six months after the fall he developed epilepsy. Ten years after when I saw him he was a victim of the disease in its worst form and exceedingly difficult to control. His cousin, the daughter of his uncle, has been an epileptic from her seventh year. In her case there is no history of injury, but in every other way her condition is similar to her cousin. As to heredity. The boy's father is a heavy secret drinker; the mother sober; one of his aunts on his mother's side, a confirmed drunkard, going on protracted "sprees" at frequent intervals. Several of the female members of the family on the mother's side and the mother's grand-uncle were also heavy drinkers. The cousin's father went on "drunks" at long intervals and when

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in liquor was a man of violent and uncontrollable temper.

Walter D. was the son of an intemperate father. When fourteen years old he developed "fainting spells," which on investigation turned out to be epilepsy. The boy responded to treatment most favorably, having had but six seizures in eleven years. In this case I found a history of alcoholism running back through four generations, all of them being on the paternal side.

Michael K. although the son of a sober father and mother, developed epilepsy at the age of five years and for twelve years after suffered from a most violent variety of the disease. In the forlorn hope of having something done for his relief he submitted to craniotomy on the advice of a local surgeon, but died an hour after the completion of the operation. In tracing back the ancestry of this boy I found the main and many of the collateral branches of the family confirmed drunkards.

All of these experiences simply indicate to me the greater importance of being well born—we should be careful in the selection of our parents, as Henry Ward Beecher was fond of saying, of being intelligently conceived by normal progenitors, and of *so* teaching the community that *they* may save their children from the baleful consequences flowing from the "insanity of rum," which often are as fraught with evil as those flowing from the insanity of mental disorder.

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EPILEPSY, ITS DEFINITION, TREATMENT, ETC.

By Dr. J. S. DeJarnette, Staunton, Va.

In discussing a subject intelligently there must be a common understanding of terms. The word "Epilepsy" literally means to seize upon, or to attack. This conveys but little idea as to the nature of the disease, as many diseases seize upon or attack the patient.

Gower says: "Epilepsy is a disease in which there are convulsions of a certain type, or sudden loss or impairment of consciousness, but in which the convulsions are not directly due to active organic brain disease, to reflex irritation, or to abnormal blood states, and in which isolated loss of consciousness is not the result of primary failure of the heart's action."

It is plain from the above statement that Dr. Gowers is attempting to tell us what epilepsy is by telling us what it is not. This, to my mind, is quite confusing and unsatisfactory. Dr. W. P. Spratling, in his excellent book on epilepsy, says: "Epilepsy is one of the most protean, variable and uncertain of all maladies common to the human race; for it is a disease which is partly mental and partly physical—more the former at one time, more the latter at another."

After quoting five definitions of epilepsy, he gives the following which he says is perhaps as satisfactory as any that can be formulated at this time: "Epilepsy is a dis-

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ease, or disorder, affecting the brain, characterized by recurrent paroxysms which are abrupt in appearance, variable in duration, but generally short, and in which there is impairment or loss of consciousness, together with impairment of motor-coordination, with or without convulsions.''

This definition takes in too many diseases that are not epilepsy, and is too generic. For instance, such a definition includes simple syncope, which recurs from time to time. Many people faint at sight of blood, or when greatly excited, etc., for this is a disorder affecting the brain, which recurs accompanied by the loss of consciousness and impairment of motor-coordination, etc. Also the convulsions of paresis, puerperal convulsions, eclampsias of children, grave hysterical attacks, convulsions of cerebro-spinal-meningitis, etc., will come under this definition.

Again, the following diseases or disorders which do not have convulsions: Vertigo, great anger, excitement or fear, short attacks of acute mania, or periodical insanity, short attacks of sleep as occur frequently in the very old, drunkenness, etc., are included in this definition, for Dr. Spratling says it is a ''disease or disorder affecting the brain, with or without convulsions.'' Clouston, Kraepelin, and De Fursac, three of the latest authors on insanity, do not attempt to define epilepsy.

While recognizing imperfections in the following, I feel that it is quite a practical definition of the disease, and hope someone will perfect it, and shorten it. It was formulated by Doctor J. W. Freed and myself: ''Epilepsy is a chronic disease, characterized by sudden, occasional recurring, and involuntary discharges, usually short in duration, from a diseased cerebral-cortex; always with

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psychical disturbance, frequently with convulsions, excluding the diseases paresis, meningitis, and short attacks of mania. The tendency is always toward dementia."

This definition does not include syncope, vertigo, short periods of sleep, and hysterical attacks, as they are not due to chronic cortical disease. Puerperal convulsions and drunkenness are due to acute systematic poisoning. The eclampsias of children are due to reflex disturbances. Paresis, meningitis and short attacks of mania have such distinctive symptons that they are not likely to be confused with epilepsy.

ETIOLOGY

I believe heredity plays a much more important part in the causation of epilepsy than Dr. Spratling and other late writers claim, which is about fifty-six per cent; for it is very difficult to obtain the correct heredity in these cases, as many families refuse to acknowledge epilepsy, or other hereditary taint, looking upon these diseases as a disgrace. Also many epileptics go through life undetected as such. For example: Cases of petit-mal when diagnosed as epilepsy are frequently a surprise to the family, who have considered these attacks to be simply dizzy, or fainting spells.

Maudsley's statement concerning insanity and crime applies well to this disease. He says: "They are as much manufactured articles as steam engines, and calico printing presses. They are neither accidents nor anomalies in the universe, but come by law and testify to causality, and it is the business of science to find out what the causes are, and by what law they work."

There are a great many theories as to the actual condition of the cortex of the brain during the convulsion. One theory is the loss of power in the nerve cells of the cortex

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on account of malformation or faulty insulation to retain accumulated nerve force, and allows it to escape at irregular and uncertain times independent of the will. Another theory is nuclear-degeneration in the cells of the second cortical layer; and another is over-production of nerve force.

I believe in every epileptic attack there is some stimulus or irritation which touches the button, so to speak, and brings on the attack, though it is often impossible to detect it. An analogous condition is seen in poisoning by strychnine, and the infection of tetanus; the barking of a dog, gently fanning the patient, or the slightest irritation to any of the sensory nerves, often brings on a convulsion. I treated a patient last year who almost invariably had a convulsion on hearing a sudden, loud noise, such as blowing of a locomotive whistle, or smacking your hands loudly close to his ears.

Dr. Lewis Bruce advances the theory that the disease is caused by some toxine developed within the system. He says: "The temperature is irregular and liable to become febrile independent of outward bodily or mental symptoms, leucocytosis is frequently raised." Haig regards the disease as due to uric acid poisoning. Krainsky injected defibrinated blood drawn from epileptic patients during a fit in the venous circulation of rabbits, and true epileptic fits resulted. He regards the toxic agents as carbamic acid and ammonia.

The blood supply of the brain is immense. The gray matter of the convolutions uses five times as much blood as the white, and needs and uses far more blood than any other tissue in the body in proportion to its bulk. The blood vessels, lymphatics, nerves, muscles, etc., are the servants of the nerve cells and not their masters, except in rare instances.

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Paralysis indicates destruction of nerve cells, while convulsions indicate activity. The disease is more liable to occur in children than in adults, because reflex action is stronger and more easily induced in them, as seen in sucking in the infant, crying, laryngismus stridulus, croup, etc. These conditions rarely occur in the adult.

Convulsions are far nearer normal action than their startling aspect suggests. In health the nerve centers are always on the alert for instantaneous, vigorous liberation of nerve force. A perception of danger induces in men and animals motor activity as intense as an epileptic fit. Once the brain cortex has discharged nerve energy without normal stimuli, a similar discharge is liable to occur again and again, as the channels of escape are made deeper and broader by each successive attack. Thus every fit is in part at least the result of those which have preceded it. Hence the cause of the first fit must be the cause of the disease. (Gowers.)

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The readiness to liberate muscular force in health continues in disease. So it shows possibly that a very slight local cause in the centers may give powerful muscular results.

EPILEPTIC PERSONALITY

The two most cardinal characteristics of the epileptic disposition are: impulsiveness and irritability. Some one has aptly said: "They have a wound-up temper." Clouston says: "Never contradict an epileptic." He is subject to terrible fits of anger for little or no cause. Other characteristics are, his liability to insanity at any time, and to do what he would not do if it were not for his disease. He is very unreasonable, has sudden changes of moods, is very religious, especially as to outward form, and has not gratitude; is untruthful, and very hopeful of recovery; a

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great lover of home; does not realize the gravity of his trouble, probably because between the paroxysms he is so near the normal man and has little or no recollection during his convulsions, or epileptic excitements, as his mind is a blank as to what has happened. Epilepsy is next to alcohol in the causation of homicides.

TREATMENT

Under the present medication of epileptics we see very little bromism in hospitals and institutions. This is due to the fact that in many of the hospitals the bromides are not used, while in others they are given with great care. I believe bromide of potash, given properly, is still the king of remedies for the disease, and I agree with Clouston when he says: "Find the dose which will not bromize, which is usually forty to seventy grains a day, in different cases, and give bromides as steadily and regularly as you give foods. Intermittent bromide treatment is of little or no use." He mentions a case in which he gave it for eighteen years with immense benefit. McPhail found that the quality of the blood improved during the treatment with bromide of potash. Clouston claims: "About one-fifth of the cases will not be benefited; four-fifths will be benefited more or less; one-half benefited very much; and one-fourth benefited so much as to be practically cured while under the bromide treatment. Its use will oftentimes save epileptics from being sent to the asylum, and any physician to an asylum who does not keep most of his epileptics continually under the influence of bromides deprives them of the best remedy we have. It makes them more tractable, and checks to a wonderful degree their impulsiveness and irritability." It should be largely diluted with water, and the patient's bowels should be opened regularly if he suffers from constipation

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caused by the drug or otherwise. Much discredit has been cast upon the bromide treatment because it has been handled so carelessly and unscientifically.

Fleichsig's treatment consists in keeping the patient in mild opium narcosis for six weeks, beginning with small amounts daily, and increasing it to fifteen grains by the end of that time, then the opium is suddenly withdrawn and 130 grains of bromide given daily for two months, then it is reduced to thirty grains per day. I have never used this treatment, as it never appealed to my judgment. Chloral is of little or no use for general treatment, but it is by far the best remedy for Status Epilepticus given in large doses, from thirty to forty grains, preferably by the mouth, in conjunction with hypodermoclysis. The chloral can be repeated in two hours if necessary, and if it cannot be given by the mouth give it by enema. I have found chloroform useful in controlling the convulsion until the chloral can act. I have used nitroglycerine with no satisfactory results. Compression of the carotids should be tried if arterial tension is very strong in status.

I have tried chloretone in a dozen cases, and in one it was more effective than any other remedy. In all the others it was worthless. Horse nettle and calabar bean have been found to be of no benefit. Dr. Peterson says simulo has no effect in most cases. Trional, antipyrine, phenacetin, and acetanilid have no beneficial influence. Zinc and copper sulphate, silver nitrate, biborate of sodium, arsenic, colchicum, are all practically useless in controlling the convulsions.

Richet and Toulouse recommend the following diet per day while giving bromide of sodium with sodium chloride eliminated from the food: Milk, thirty-five ounces; butcher's meat, ten ounces; potatoes, ten ounces; flour, seven

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ounces; sugar, one and one-third ounces; butter, one and one-half ounces; and water in large quantities.

Serum treatment does not give much encouragement at present, though I believe the opsonic treatment offers some hope. Correcting vision and muscular unbalance of the eye has but little effect in the disease. It is a strange fact that inter-current diseases such as pneumonia, fever, fractures, etc., often diminish the convulsions to a great extent.

Almost every remedy in the materia medica has been used for epilepsy, but those enumerated above are the chief ones that have been of service up to this time. Some recommend the salts treatment, in which the patient is given a dose of epsom salts twice a week whether he is constipated or not. This does not appear scientific to me, and has a tendency to lower the vitality of the patient if kept up any length of time.

Marriage of epileptics and other mental defectives should be prevented by law.

THE COLONY TREATMENT

The colony plan has been gradually evolved, as it has been found necessary to have absolute control of the patient as to his hours of employment, recreation, sleep, diet, education and habits. By the colony plan the patient is constantly under the care and supervision of physicians skilled in the treatment of the disease, who have authority to see that their rules and regulations are carried out. This is practically impossible at home.

The patients are taught self control and their associations are made as congenial as possible. They make their own friends when brought together and stay together, and in many instances almost inseparable. By having several cottages in the Colony they can be classified very easily.

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All who are capable should be given some work to do. They can do such work as weeding the garden, making bricks, using a pick, pushing a wheelbarrow, and in fact almost any occupation that does not require handling a team, edged tools, or working in high places.

The chronic insane, demented and feeble-minded epileptics should be put in large buildings, as it is not necessary to classify them so carefully, because they need only custodial care. All who are mentally capable should be encouraged to indulge in games and healthy out-door recreation, with short hours for work. One epileptic should never work alone, two or more should always be together for mutual protection in case of a convulsion. His education should consist of physical and manual instruction.

DIET

Epileptic diet should be good, nutritious, easily digested food. The majority of them can eat meat once a day without bad results. He should eat regularly and not overcrowd his stomach at any time. On account of his severe muscular storms, the epileptic requires more food than the average man, and has a voracious appetite. He should have an hour for his meals, and be taught to eat slowly. Fried food should not be given; the best drink is milk. All ripe fruit and vegetables, I think, go to make up the proper diet, and he should be moderate in all things.

SURGICAL TREATMENT

I do not believe that petit mal, hystero epilepsy, convulsive storms, and status epilepticus should be treated surgically. The operations for grand mal that have come under my observation have been very unsatisfactory. Whenever a case has a strong epileptic heredity an operation should be undertaken with great caution. Of course

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all depressed fractures, or clots on the brain, should be promptly relieved at the time of the injury, whether the patient is epileptic or not. Trephining in epilepsy is not nearly so common now as it has been.

PROGNOSIS

The prognosis of this disease is always grave as to recovery, though I believe most cases, if not demented, can be benefited. Sixty per cent die as a result of their seizures (Worcester), and, as Clouston says: "Twenty per cent can be made useful citizens." Gowers estimates that about twelve per cent of epileptics become insane. The most hopeful authorities estimate from five to ten per cent curable. This must be based upon the treatment of the non-insane epileptic, for there are very few recoveries among the insane epileptics. The difficulties in the treatment of the epileptic is the same now as in the days of Christ, when "There came to Him a certain man kneeling down to Him and saying: 'Lord, have mercy on my son, for he is a lunatic and sore vexed; for oftimes he falleth into the fire, and oft into the water, and I have brought him to Thy disciples, and they could not cure him.' Then came the disciples to Jesus apart and said: 'Why could we not cast him out?' and Jesus said unto them: 'because of your unbelief.'" This unbelief in the curability of epilepsy still exists, and has discouraged to a great extent scientific investigation and experiment as to the cause and cure of this dread disease. If our faith be great, good results will attend our patient labors;

> "The heights by great men reached and kept,
> Were not attained by sudden flight;
> But they, while their companions slept,
> Were toiling upward in the night."

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A CONSIDERATION OF HEREDITY AS A FACTOR IN THE CAUSATION OF EPILEPSY

By Morgan B. Hodskins, M. D., Palmer, Mass.

In looking over the statistics on heredity in this disease by the different authors, one is struck by their wide variation.

The following table gives some of the recent statistics :—

AUTHOR	TOTAL CASES	PER CENT WITH HEREDITY HISTORY.
Echeverria	306	25
E. Reynolds	38	31
Moreau	364	32
Aronsohn	508	32
O. Berger	71	32.3
Binswanger	150	36.3
Gowers	2400	40
Spratling	1070	56
Dejerine	350	66.8

On investigating this discrepancy, it is found that the authors who have the greatest number of cases with a history of heredity include those patients whose ancestors have suffered with such diseases as rheumatism, tuberculosis, etc., and at the same time include diseased states in the cousins, apparently forgetting that here we have the introduction of an entirely new strain of blood.

Having these facts and knowing the almost universal tendency to ascribe nervous diseases, and particularly epilepsy, to heredity, we should leave out the purely

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accidental and only consider those cases as showing heredity whose direct ancestors show some nervous or mental disease, such as epilepsy, insanity, migraine, organic nervous disease, etc.

An investigation on this line of twelve hundred cases of epilepsy, which have been admitted at the Massachusetts Hospital for Epileptics in the past nine years, yields the following information:

Of the 1,200 cases, 700 were males and 500 females.

Of the whole number (1,200) 173 or 14.41 per cent gave a history of heredity.

Of the males (700) 86 or 12.28 per cent showed heredity.

Of the females (500) 87 or 17.4 gave a history of heredity.

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A CASE OF EPILEPSY OF THE FAMILY TYPE

By Wharton Sinkler, M. D., Philadelphia

There is a general opinion that direct heredity is the most important factor in epilepsy, and this is true to a certain extent, but I believe that careful investigation of all the cases recorded will show that the number of epileptics in which a parent or gandparent has suffered from the same disease is comparatively small. Spratling —"Epilepsy and Its Treatment," page 64, finds that of 1,070 cases which came under his observation (660 men and 410 women,) fifteen per cent of the men and seventeen per cent of the women had epilepsy because of the same disease in the parents. Dejerine, quoted by William Aldren Turner—"Epilepsy, A Study of the Idiopathic Disease," London, Macmillan & Co., 1907, gives twenty-one and two-tenths per cent of epileptics with a direct inheritance of the disease. Binswanger found eleven per cent, and Doran nineteen and three-tenths per cent of their cases with a direct heredity of epilepsy. Gowers states that of 2,400 cases which were observed by him forty per cent had a hereditary history, but he probably included in his heredity, not only epilepsy but alcoholism, insanity and other neuroses. Of 1,124 cases which have been treated at the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases, in 157 cases or thirteen and ninety-six hundredths per cent, there was

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traced an hereditary cause. This heredity included epilepsy in the parents or grandparents, brothers and sisters, or in an uncle or aunt or first cousins. If we take the cases in which only the parents were affected with the same disease, the percentage would not be more than five per cent or six per cent. Very careful inquiry was made in regard to this point in taking the histories of the cases, and even allowing to deception either through ignorance or intention, the percentage could not be much greater than this.

Other neuroses no doubt play an important part in the production of epilepsy. Alcoholism in the parents is probably one of the most potent causes. Dejerine gives alcoholism as an hereditary factor in fifty-one and six-tenths per cent of his cases. Binswanger gives twenty-two per cent, Spratling fourteen per cent and Doran twenty-one and six-tenths per cent of their cases as having an alcoholic heredity. In 951 cases at the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases examined in regard to an alcoholic heredity, there were one hundred and thirty or thirteen and sixty-seven hundredths per cent in which one parent or the other was alcoholic.

The family tendency in some cases is very strong. Gowers—"Epilepsy and Other Convulsive Diseases," page 8—cites one instance in which a patient's mother, maternal aunt, two uncles and cousin were epileptic. In another of his cases there were fourteen members of the family affected. In a third, eleven, and in another case the patient's father, two of the father's brothers and three of his sisters were epileptic.

The following case shows distinctly a tendency to the disease which has extended through several branches of

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the patient's immediate family. It is suggestive of some of the family neuroses, like Friedrich's ataxia, family dystrophies, or Huntingdon's chorea.

C. B. H. Aged 26. Consulted me September 14, 1907. He has always been strictly temperate and denies any venereal disease. He says that he has always had excellent health with the exception of the trouble for which he consulted me.

His father's father and all other paternal antecedents as far as he knows, were perfectly healthy and normal. His mother's father drank a good deal and her mother's father was alcoholic. The father's mother had epilepsy, but nothing is known of her relatives. As far as the patient knows she had only one attack. He admits, however, that he is not well informed on the subject. She was of sound mind and died young, at about the age of thirty-five. His father had been married twice and has had a large number of children—about fifteen. He was entirely free from epilepsy. One brother, that is an uncle of the patient, had epilepsy quite violently, often having six or eight fits in one night, but he recovered from the attacks and has now had none for about thirty years. None of his children have been afflicted. Three sisters of the father were married. None had epilepsy, but of one, two children became epileptic, one of whom was insane and died in this condition. Each of the other sisters had one child who was epileptic. The patient himself has had two brothers and two sisters who had epilepsy. One brother and two sisters of these became insane and died in an asylum. One brother, now aged nineteen, had epileptic attacks for several years, but he has now had none for three years. So that in this and the two preceding generations there have been eleven epileptics.

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About eight years ago, when the patient was sixteen years of age, he had an epileptic seizure at night. Since that time he has had attacks recurring every three or four weeks. They generally occur at night, but for the past year there have been more in the day than previously. The longest interval that he has had since the attacks began, has been three months. The attacks have usually come on at night, but the last two or three have been in the day. On June 11th he had an attack at night in his sleep and the next morning soon after going to work he had another attack. It is only occasionally that he has had two attacks so close together. He does not bite his tongue, but occasionally bites the inside of the lower lip. He has broken his upper teeth in falling, but has done no other injury to himself. His wife states that in an attack his eyes are first staring, and then there is a general convulsion, which lasts for a few minutes; then he sleeps heavily for two hours. If the attack occurs at night, he sleeps very profoundly the remainder of the night. His memory is unimpaired, except that just after an attack he is very forgetful. About once a month he has an attack of petit mal. He becomes slightly confused for a few moments and then seems as well as ever. He works for the Southern Express Company—does office work and is very active and capable. He is a strong, well built man. He has the appearance of good health and is bright and intelligent. His heart is normal; his pulse is rather sclerotic. There is no kidney disease. He has taken bromides in doses of from ten to thirty grains three times a day, of the bromide of potassium, without apparently any marked effect.

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THE UTILIZATION OF EPILEPTIC LABOR

By H. M. Weeks, M. D., Superintendent New Jersey State Village for Epileptics, Skillman, N. J.

One of the essential needs for the establishment of colonies for the care and treatment of Epileptics is to provide regular and systematic employment for the patients, primarily for their betterment and, incidentally, to minimize the cost of their maintenance.

That employment is beneficial to the patient when wisely selected and systematically engaged in, there can be no doubt; and that it is more or less profitable to the institution must also be admitted.

The patient benefits hygienically, therapeutically and morally. The institution profits economically by the reduction in the cost of maintenance in proportion to the earning capacity of those employed.

The patient when regularly employed, is taken out of his self-consciousness, his mind is attracted to things that are conducive to a healthy mind, and the muscular exercise required assists nature to get rid of those products of metabolism that are detrimental to both mind and body. Epileptics are naturally hearty eaters, and in many cases gormands, the waste products are therefore enormous, and unless gotten rid of by a proper amount of muscular exertion, his epilepsy becomes aggravated, and his mind and body rapidly deteriorate.

Manual labor is unquestionably the best and most satis-

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factory method of securing the desired muscular exercise. The physical and mental condition improves under the stimulus, and with a better mind and body his epilepsy is certain to improve, and sooner or later he begins to realize that he is of some use in the world and that the more he does toward his self support, the less of a pauper he is; and thus stimulated and encouraged he becomes more self-reliant and more self-respected and his moral tone is elevated.

The institution profits by the labor performed by the patients to a greater or less degree as they are of a selected class or composed of all classes, as is usually the case in a colony. It has been claimed that the colonies could and should be self-supporting, "a consummation devoutly to be wished."

If patients could be selected with reference to their mental and physical condition, frequency of attacks, etc., a colony might be made more nearly self-supporting; but, even with the best class of patients—none of whom begin to equal a normal person in earning capacity—the need for proper supervision and directing of their labor requires so expensive a force that, added to their limited earning capacity, reduces the value of the results to such an extent as to nearly equal the cost of competent hired help.

Few epileptics capable and able to make a living outside are induced to go into an institution. It is only when his labor becomes non-supporting and when he can no longer obtain employment outside, that he will give up his liberty.

In an institution where all grades are gathered, and only about one-half or two-thirds, at most, are capable of doing any work at all, and few, if any, of those equal in earning capacity to more than sixty per cent of a normal

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person, it would seem logical to conclude that an institution cannot become self-supporting.

However, we should earnestly endeavor to attain that ideal as nearly as possible by utilizing the labor of the epileptic to the best possible advantage, not only for his personal benefit but to minimize the cost of his maintenance. The matter of utilizing the labor of patients should be carefully considered in the selection of a location for a colony. It is conceded that out-door employment is best suited to the requirements of epileptics, therefore, a site should be chosen that will afford the greatest facilities for such employment.

Agriculture unquestionably offers the largest and most useful and beneficial field for the employment of the colonists. The farm affords opportunity for a certain class of inmates to be employed regularly the year round in caring for poultry, swine, and assisting in the dairy and about the buildings, and for another class during certain seasons. There is much work to be done in ordinary plain farming that cannot be safely performed by the epileptic. Our experience has been that it is more profitable to hire a sufficient number of farm hands to insure the operating of the farm, and to supply the necessary extra help, required at times, by patients.

Where the soil is adapted to the raising of vegetables, truck, berries and small fruit, better results will be obtained for both patients and the institution if more attention is paid to this department than to the rotation of crops on the farm. Patients can be more regularly and systematically employed, and besides, much of this work can be done by female as well as male patients.

The products of the gardens, besides supplying all that is required for consumption in the institution, fresh,

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canned and preserved, also yields a considerable income if marketed. Asparagus and celery are very profitable crops, and are easily cared for by the patients.

The early establishment of nurseries of shade and ornamental trees and shrubs affords suitable employment for a large number of male patients, and supplies the means of providing shade about the cottages and ornamenting the grounds at a nominal cost.

Should the tract of land purchased for the location of a colony contain stone suitable for building or road construction, or a deposit of clay or gravel, most profitable industries can be carried on by utilizing the labor of the patients in manufacturing brick, quarrying stone for buildings and road making, or in building gravel roads and walks. Any one or all of the above mentioned industries can be carried on with a minimum amount of skilled supervision; hence the cost is nominal compared to the production.

Climatic conditions will not admit of out-door employment all the year, and besides there are many patients who are physically and otherwise unsuited for out-door work.

Every patient capable of understanding should be taught and encouraged to become a producer and thus contribute to his own and the colony's support.

Both male and female patients must be employed in the various departments of housekeeping: men in the houses for men and women in their homes. The women should do all the laundry work, sewing and mending of clothing for the entire institution, while the men make and repair the shoes, men's clothing, mattresses, repair furniture, make brooms, brushes, mats, weave carpets and rugs. Both men and women can be employed during the season

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of canning and preserving fruits and vegetables. Thus all are factors in making a colony self-supporting as nearly as it is possible to do so.

While it is rarely possible to develop original or skilled artisans among the epileptics, it is possible to accomplish astonishing results along many lines by patient, persevering, painstaking and encouraging training. The patient should go about his daily work cheerfully and not approach it as a task set for him. His adaptability and inclination should be studied, and when possible, congenial employment should be selected, and his training should be under the supervision of those actuated by higher and nobler impulses than those of a keeper or overseer whose only ambition is to put in time and draw a monthly wage.

Better and better will be the results as the colony system is more thoroughly understood and the young are committed to our care.

The education and training should begin as early as possible, before the habit of indolence has been formed, and before the patient has met the discouragements the world offers on account of his disease, and before he has become contaminated by evil associations and vice, often due to enforced idleness before entering a colony.

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COLONY LIFE OF AN EPILEPTIC, SOCIAL AND RELIGIOUS

By Rev. J. Duncan MacNair, Sonyea, N. Y., Protestant Chaplain, The Craig Colony for Epileptics

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The topic of the social and religious life of an epileptic on the colony plan is a matter of not a little speculation, but enough is known to lay down general rules, which if deviated from bring ruin to the patient. The discussion will only cover points which are at once practical and at the same time easily carried into effect. I will not presume to dictate to any present nor to be dogmatic in the position which I take, but still, from constant observation of colony life for the past fifteen months, certain phases have impressed me as to their importance.

The epileptic is moody, impulsive, emotional and often very irrational, thus making it very difficult to manage him; but these different temperaments may be modified by proper care and supervision. The majority of epileptics have been considered by their parents and friends as closet skeletons, hence these unfortunates (being the most so of any class of invalids in existence) have been deprived of social and religious privileges, which are the only pleasures and comforts to be gotten by any individual in life. To give an example: on my former charge there was an epileptic, a sister of one of the church deacons, whom in my year and a quarter's stay there I had never seen,

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although I had called at that house on an average of once a month, and still further no mention was ever made of her by them to me, and for nearly a year I knew nought of her existence until a neighbor told me about her. This is the case of the majority, although I am happy to say that there are some exceptions. Thus coming, as they do, from a literal bondage to a life of practical freedom, the problem of how they are to be helped the most and the greatest good done is hard to solve.

The social life of epileptics may be considered under four heads: namely, work, recreation, the intermingling of the sexes and the laws governing them. Work is perhaps the most essential, as it is the means of livelihood and a great help in lessening their afflictions. They are not averse to labor although they at times must be urged to do so, and in this respect are not dissimilar to a great number of normal persons. To give them employment, something to take their minds off their trouble is necessary. It is not too much to say that where those who are physically able are employed they are better, have a less number of attacks and are happier. This is not hard to account for, as work, where congenial, concentrates the mind, exercises the body, makes the food taste more wholesome, fatigues the body so as to make them rest better, all of which are conducive to the obtaining of the best results in any individual and no less so in an epileptic. In the last sense we spoke of the work being agreeable, and in this one will find that it is not always easy to give them something to do that is pleasing, but the general rule is to divide the labor as equally as possible and to give the different parts to those best suited to do them. There are as many different dispositions among them as there are persons and some have a different one for every

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hour of the day. Outdoor work is the best suited to the disease as it furnishes exercise in the open air.

After work comes play. Recreation is simply re-creation. One would naturally suppose that the epileptic would want to take an active part in sports, but the following statistics will prove that the reverse is true. Among six hundred male patients on Craig Colony not more than ten per cent will take an active part in any game, but all are willing to watch when one is in progress. Among five hundred women only one was found who knew anything of lawn tennis and three were willing to learn; about sixteen would play croquet; less than ten were found that cared enough for flowers to cultivate them; ten knew how to skate and fifteen more wanted to learn; about thirty per cent cared to take walks and the rest, while being willing to be entertained, were unwilling to do anything. Thus the recreation proposition is difficult to manage. This listlessness and disinterestedness is an indication of the disease. It saps the energy and takes away ambition. To excite interest and to make it a pleasure for the majority is almost impossible. To turn their thoughts away from themselves and to concentrate them on something else is the object to be obtained. Twenty-five per cent are fond of reading, five per cent can play some musical instrument, and fifteen per cent can sing. Last year we endeavored to form walking clubs, and although several attempts were made to organize we were unable to interest the patients and even those who liked to walk preferred to go by themselves or in company with a friend. From the foregoing, certain conclusions can be drawn. The recreation must be suited to the individual but constant efforts must be made to turn the desires of the epileptic toward such forms of relaxation as will develop

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his mind and his body, at least to keep them in as good condition as possible, and this can be done, as in work, to the best advantage by out-of-door sports.

It is often asked if it is well for epileptics, male and female, to intermingle. The only answer that can honestly be given to this is "no." The reason for this emphatic and curt answer is that they are inclined to be very sensual and many that have the disease are given to perversion, and to allow them to be much together only serves to excite those passions. Then again it is not right to allow them to marry, for to do so means more epileptics brought into existence. There ought to be a law passed in every State in the Union and by all civilized countries to the effect that none should be allowed to marry. We would not go so far as a former chaplain went in saying that often when they congregate for religious service or an entertainment the sight of one of the opposite sex brought on an attack, but we would say from a close study that to allow them to intermingle promiscuously makes them almost unmanageable. However, there are a few that do act like normal individuals when allowed those privileges.

The laws governing them on a colony should be strict enough to keep them regular in everything that they do, whether at work or at play. As a class, it is impossible to make them self-governing as is done in some social settlements. It is often said by those who have direct charge over them, that to give them one privilege is to give them a score. This in our case we have found true, for if ever there is a persistent bore the epileptic is one. This is due to the condition of his disease, for he yearns for sympathy, kindness and friendliness and when these are shown he takes it for granted that he is a privileged

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character to the one showing these qualities. This is where the difficulty lies with the employees, for to know just when to be kind, sympathetic, considerate, and when to be stern and unyielding in governing is hard to decide. The code of laws drawn up for Colony use must be such as will keep the colonist regular in his habits, not too severe, but strict enough to have them obeyed and carried out in an impartial and just manner.

We must now consider the religious life of epileptics. There are some people who think that any kind of a church service will do, but in this they are mistaken, for there is no class of people that need more thought and care in religious training than they. They are inclined to be very religious, in fact some are fanatical in their religious thought; but their spirituality is not deeply rooted, and they are apt to be swayed by the prevailing emotion. They mean to do what is right, but the will power is weak and the judgment is impaired so that they are unable to make clear distinctions between right and wrong conduct. This makes it very perplexing to teach them right living in a practical manner. The services should always be unemotional, for nothing will hinder the work so much as to have their emotions stirred, but should be interesting enough to hold the attention. In our meetings during the past year, seizures while the sermon was being delivered were rare, but during the other parts of the service they occurred quite frequently and as many as ten have been noted in the first twenty-five minutes. Thus it seems only fair to deduce that ninety per cent of the seizures occur during the ritualistic part (especially where they take no part) and ten per cent during the sermon. This can be accounted for by the theory that they are more directly concerned in the

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preaching, as it is more or less directed to them and thus holds their attention, while during the other part they are listless and apt to be inattentive.

The concentration of the mind evidently retards and sometimes wards off an attack, especially if it be a psychic or a petit mal. Further it may be noted that our evening meetings, which consist largely of singing with a sermonette, are often without a single attack, still further proving that intense interest has a beneficial effect upon the epileptic, for if it does not actually ward off a seizure it at least retards it. Therefore the kind of service that is needed is one that is unemotional, thoroughly interesting, with plenty of good singing and music, and simple enough so that the one with a weak mind may derive some good from it, and complex enough to have instructed the brightest in the ways of God and morality. The sermons should be directed to the benefit derived from living religious and moral lives.

Christian work bears much fruit. The patient instead of becoming morose, despondent and sad learns to see another side of life. He is taught that there is a kind, loving father who is interested and cares for him; that, although he may never be cured of his malady, yet God has created him for a purpose and that if he is true to that Father, He will reward him with a better and happier life at some future time. This kind of teaching makes him hopeful, happy and much easier to get along with. Keep religious privileges from him and you will find him much harder to control; but as soon as given him the tide turns, for he is filled with new desires and new ambitions, a desire to do right and an ambition to be pure in heart and mind. No matter how far below the standard he may fall, he usually strives to reach it, and after all it is this

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striving that counts in early life. There is abundant proof for this statement in that nearly every supervisor and nurse has commented on the difference in the dispositions of the patients with and without religious services.

In conclusion, it might be well to state that all the statistics have been made from The Craig Colony, also that the observations were made there. Some statements in this paper may not apply to all epileptics outside of a colony, but they will be found correct and applicable for patients whose disease has taken on an advanced and aggravated form, and of those who have become State charges partially or wholly.

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ACUTE PULMONARY EDEMA AS A COMPLICATION OF EPILEPTIC SEIZURES

By William T. Shanahan, M. D., First Assistant Physician, The Craig Colony for Epileptics, Sonyea, N. Y.

The complication of a grand mal seizure mentioned in this short paper is that of a pulmonary edema which is sudden in onset and produces a serious condition in all instances and in some cases a fatal termination. In some cases this edema is recurrent. This state is one to which little or no attention has been called in treatises on epilepsy, although seen fairly frequently.

Riesman, in an excellent article in the American Journal of Medical Sciences, January, 1907, has given a resume of the literature on the subject of pulmonary edema and reports six cases of an acute recurrent form.

He mentions this edema as being associated with arteric sclerosis, Bright's disease, heart disease, angina pectoris, asthma, acute infectious diseases, pregnancy, paracentesis of thorax and abdomen, angeio neurotic edema and, finally, with obscure conditions of questionable causal relationship, such as hysteria, etc.

In every grand mal and in many petit mal seizures there is a more or less marked increase in the secretion of mucus along the respiratory passages, with the subsequent frothing at the mouth which is commonly seen in such patients. In some cases there may be no actual increase, but simply an accumulation in the mouth of the normal

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amount which is then forced from between the lips during the accentuated respiratory air currents which accompany the stertorous type of breathing.

In a few cases this great outpouring of mucus becomes so marked that the patient is in actual danger of being drowned in his own secretion, this acute process producing a marked pulmonary edema. This acute edema is not meant to be confounded with the frequently occurring terminal edema seen in many epileptics, as well as those suffering from the many other diseased states which have already been mentioned. In a large proportion of the autopsies on patients who had died at the Colony, a terminal pulmonary edema has been found.

The patients observed have been in fairly good physical condition preceding the occurrences of one or more grand mal seizures. During the stertorous period of the isolated seizure, or perhaps the second or third in a series of seizures, there suddenly appears an outpouring of mucus, perhaps blood-tinged, from the mouth and nose with a marked cyanosis and dyspnoea occurring concomitantly.

Etiology.—Some of these cases occurred in patients who had an affection, which in all probability acted as a predisposing cause, but would never have been sufficient to have brought about the edema unless the circulatory changes accompanying the epileptic seizure had been added to it. In the eleven cases reported, seven recovered and four died. In three cases the condition recurred, and in one of them the second period observed was fatal.

Dr. Wm. H. Welch's theory of the cause of pulmonary edema is as follows:

> "A disproportion between the working power of the left ventricle and of the right ventricle of such character that, the resistance remaining the same, the

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left heart is unable to expel in a unit of time the same quantity of blood as the right heart."

An enfeebled action of the left ventricle, rather than a spasmodic action, is likely to be the cause of this disproportion under the conditions in which pulmonary edema ordinarily occurs. As a consequence of this, there is soon a large excess of blood in the pulmonary vessels. The edema is one usually associated with venous hyperemia. An increase of the intra capillary pressure is brought about in the lungs and perhaps a decrease of the extra capillary pressure, or both. The asphyxia accompanying the seizure is caused by the local stagnation of the blood stream and in turn causes an increase in the permeability of the endothelial wall of the capillaries of the lung.

Treatment.—The most important procedure has been found to be a change of posture, so as to aid in draining the respiratory passages of the great excess of mucus. If necessary, turn the patient over the side of the bed, removing all mucus from the mouth, nares and pharynx. Open the mouth with a gag or with the fingers protected by a towel. In addition, much benefit has been obtained by dry cupping, and the use of atropine and strychnine hypodermically. Venesection is of value in full-blooded patients. Adrenalin was used without apparent result.

Case I. M. B. T. Female. Admitted to The Craig Colony May 22, 1899. Age, 27 years. Onset of epilepsy at seven years. Has both petit mal and grand mal seizures. Well nourished woman. At time of admission physical examination revealed a mitral murmur.

July 13, 1903. She had a grand mal seizure, following which a marked acute pulmonary edema occurred. A venesection was done and patient quickly improved. Recovery uneventful.

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December 6, 1906. Slight bronchitis had occurred at intervals during past year. General health had been good, so that she worked in laundry regularly. At 1:15 a. m. she had a severe seizure, which was followed by a marked cyanosis of face, dyspnoea and elevation of temperature, 103 degrees F. Examination of chest showed numerous dry rales and some moist rales. There was some bloody saliva and mucus expectorated from time to time. Great restlessness and fear of death. She was turned on her side with head hanging over edge of bed so that fluid would drain from air passages. Given atropine grain one-sixtieth hypodermically and dry cupped over the entire chest. In half an hour patient was quieter and dyspnoea much less marked. Pulse was of good volume but not of particularly high tension. Moist rales were present for three days, after which she returned to her usual state of health.

Case II. H. J. L. Male. Admitted April 9, 1898. Age thirty-six years. Evidences of beginning pulmonary tuberculosis, but no heart lesion was present. Had both grand mal and petit mal seizures.

October 4, 1906. Following serial attacks, this patient developed an acute pulmonary edema. Loud moist rales were heard over entire chest, and mouth filled with frothy mucus. Pulse is full and strong and respiration noisy. Examination of heart unsatisfactory, owing to noisy breathing, but there is apparently a murmur accompanying second sound at apex.

October 5, 1906. Condition became worse; seizures recurred until death occurred at 1:45 p. m. Temperature was not above 100.8 degrees F. No autopsy was permitted.

Case III. Lillian S. Age, twenty-six years. Onset

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of epilepsy at thirteen years. Both grand mal and petit mal. Patient well nourished. No heart lesion and kidneys normal.

June 9, 1906. This evening patient had one grand mal seizure, following which she had a very marked pulmonary edema. Profuse blood-tinged frothy expectoration. Patient received atropine grain one-fiftieth, strychnine grain one-thirtieth hypodermically, and was dry cupped over chest. Temperature 100 degrees F., pulse 120, respiration 68. Foot of bed was raised. Condition improved rapidly and patient was in usual condition in thirty-six hours.

Case IV. Chas. W. J. Admitted to The Craig Colony March 1, 1901. Age, nine years. Seizures grand mal in type. No heart lesion admitted.

September 10, 1906. Following serial attacks, he had marked pulmonary edema. Entire chest dull on percussion and coarse moist rales present in large numbers. Given atropine and whiskey hypodermically, and mustard paste applied to chest. Temperature 105 degrees F., pulse 150, respiration 60. Temperature lowered to 103 degrees F. by alcohol sponge. Three hours later temperature was 105.4 degrees F., pulse 140, and barely perceptible at wrist. Edema slightly cleared up. Frequent seizures continued. Patient rapidly became weaker and death occurred six hours later. Autopsy by Dr. J. F. Munson showed lungs very moist and on section surface moist and somewhat foamy. Small amount of fluid in each pleural cavity. Heart weighed 165 grammes. Left ventricle firmly contracted. Muscle felt firm and good. Mitral valve admitted forefinger. Mixed clot under mitral valve flapped and extended up into auricle, making a cast of their cavities. Valve edges were thickened but not hardened.

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Pulmonary artery admitted thumb with difficulty. Flaps were normal. Thymus persistent.

Case V. H. M. C. Female. Admitted July 1, 1902. Type, grand mal and petit mal. Onset in early childhood. Note on admission read first sound of heart was not clear.

January 31, 1906. This patient had some severe seizures early today and soon developed a pulmonary edema. Temperature (rectal) at 7 A. M., 101 degrees F., pulse 100, respiration 20. At 10 A. M. she was dry cupped on back along both sides of vertebral column. Given atropine grain one one-hundredth every three hours. Large and small mucous rales can be heard over entire chest, but especially posteriorly over left lung. Palpation readily shows fluid in air passages. Because of large amount of adipose tissue, percussion is unsatisfactory. Heart sounds rather indistinct but appeared normal. Pulse was of fair volume. Patient was partially conscious in interval between seizures and took some milk by mouth. Pupils were even and not dilated. No evidence of paralysis. Breathing stertorous. At 2 p. m. temperature 99 degrees, F., pulse 120, respiration 40. At 2:45 p. m. grand mal seizure. At 4 p. m. rales seemed more numerous. She had another severe seizure at 5:35 p. m., and was much worse thereafter. Heart sounds were quite good at 7 p. m., as was also pulse. Temperature (rectal), 101.5 degrees F., pulse 132, respiration 40. Condition grew worse and death occurred at 8 p. m. Considerable cyanosis and laryngeal rales were present for some time before death. Autopsy was not permitted.

Case VI. B. M. J. Female. Admitted December 18, 1902. Age, eleven years. Type of seizures, grand mal. Onset at three and one-half years. Second sound accentuated. Urine normal.

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January 18, 1906. At 5:45 p. m. yesterday this patient began having severe seizures in rapid succession. She had just finished her supper and had appeared as usual. At 6 p. m. rectal temperature was 99.4 degrees F., pulse 118, respiration 34. Patient was unconscious and facial muscles were in clonic convulsions with no interval free from convulsions. High cleansing enema ordered to be followed by enema of chloral hydrate grains XX, and potassium bromide grains XXX. Seizures were so severe that after fourteenth, physician administered chloroform by inhalation until seizures stopped. There were marked stertor and increase of mucus in upper respiratory passages. After seizures stopped the mucus was so profuse that it poured through nose, patient became markedly cyanotic and respiration irregular and shallow. She was turned on her side and her head allowed to hang over the edge of the bed with her face down. Her mouth was pried open with a spoon and held so with the handle of a tablespoon; then the tongue was grasped with a towel and pulled out far enough to permit air to pass through larynx. Mucus was expelled from nose and mouth. Her condition began to improve and she began to return to consciousness. Pulse continued of good volume throughout. At 7 p. m., rectal temperature was 98.6 degrees F., pulse 120. At 10 p. m., she was restless, tossing about bed. With this exception she appeared to be in her usual condition. The following morning she was up to breakfast and about as usual.

April 1, 1906. No prostration followed another series of seizures shortly after the one described. Physical condition was good.

July 31, 1907. The patient had eleven grand mal seizures between 7 a. m. and 8:35 p. m. The last seven

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occurred in rapid succession between 8 p. m. and 8:35 p. m. Given chloral hydrate grains XX, potassium bromide grains XXX, by enema. Foot of bed elevated six inches. 9 a. m., atropine sulphate grain one-fiftieth hypodermically. Mustard paste applied to chest for twenty minutes. Following the last seizure patient developed a slight pulmonary edema. Examination of chest showed slight impairment of resonance over entire pulmonary area. Rhonchal fremitus over same area. Scattered large moist rales over both lungs but most numerous over upper and middle lobes of left lung anteriorly. Pupils dilated. Patellar reflexes very slight. The remaining reflexes were unobtainable. Patient unconscious and slightly cyanotic. Pulse at 11 p. m. 100, regular and full; temperature 100 degrees, respiration 24. Normal saline solution, one pint, given by rectum and repeated every three hours until patient returned to usual condition.

August 1, 1907. Patient is bright and wishes to get up. Examination of chest negative, except a few scattered moist rales over left middle lobe anteriorly and in left axilla. 8 a. m., temperature 100 degrees, pulse 86, respiration 20.

August 2, 1907. Examination of lungs negative and patient appeared in her usual condition.

Case VII. C. P. Male. Age on admission, April 4, 1903, was twenty-four years. Onset at eight years; grand mal and petit mal. Patient was well nourished, and heart and kidneys were normal.

November 14, 1905. This patient has had quite frequent seizures for past few days, having had thirteen in three days, and this morning he had eleven. He was mentally disturbed since the day before, was constantly muttering to himself, and frequently cried out. His temperature was 102 degrees. He was transferred to the hos-

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pital. Examination showed both lungs filled with loud moist rales, and some dullness was noted over left lung in front. Tongue was coated with heavy brownish fur. There was some congestion of face and neck, which increased during the evening. Respirations were labored and noisy, abdominal muscles being brought into use. Mouth was filled with a thick viscid mucus which had to be cleansed quite often.

November 15, 1905. He was disturbed during early part of night, but after 10 p. m. was quiet. Temperature rose last evening to 102 degrees F., but this morning was 100 degrees F. Free perspiration last night, and patient could not be aroused. Saline infusion into both mammary regions this morning. Pulse was rapid and feeble, the respirations being less frequent and more shallow, and patient died at 10:50 a. m.

Case VIII. I. M. B., admitted June 21, 1905; age, sixteen years. Onset at six years. Heart and kidneys were normal.

October 1, 1905. This patient had four mild seizures yesterday and complained of pain in her abdomen. At about 4:45 p. m. she passed into a condition of status epilepticus, seizures beginning by head being turned to right. When seen by physician she was having a general clonic convulsion, was markedly cyanosed, breathing stertorous, with an unusual amount of mucus in the upper respiratory passages; bathed in a profuse perspiration, pupils small and no reaction to light, corneal reflex absent, reflexes absent in extremities; pulse 160; rectal temperature at that time was 102.6 degrees F. This morning she was in her usual condition. After convulsion she had a slight lateral nystagmus. Right pupil was considerably larger than left, but both responded quickly to light.

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March 12, 1906. At about 10 p. m. yesterday, night nurse found this patient in a condition as if she had had a severe seizure. Patients in same room said she had convulsed for half an hour. She was breathing in a stertorous manner, with considerable mucus in upper respiratory tract. Pupils were moderately dilated. Pulse 150 per minute, but of good volume; rectal temperature 103 degrees F. Patient semi-conscious and very restless. Knee jerks, wrist jerks, and elbow jerks were active; no ankle clonus; Babinski reflex present on both sides. At 11 p. m. temperature had dropped over two degrees. At 7 a. m. temperature 100.4 degrees F. Tongue lacerated. Patient was pale, but otherwise in about her usual condition. Later in the year she had a similar condition develop following seizures. Recovery.

Case IX. E. F., female; admitted March 24, 1904; age, seventeen years. Grand mal. Onset at one year. Accentuation of second pulmonic sound. Urine normal.

May 5, 1905. This patient had a grand mal seizure last evening. After the attack she developed a moderate hemoptysis which gradually became less marked. Ice was applied over her chest and she was given fragments of ice to place in her mouth. Morphine, grain one-fourth, and atropine, grain one one-hundred and fiftieth, were given hypodermatically. Pulse was 120. This morning she was quite comfortable. Temperature 98.8 degrees F., pulse 112, respiration 35. She had very large tonsils and probably adenoid in nasopharynx. This morning an examination of the chest revealed many coarse rales over right lung, especially along border of ensiform appendix.

Case X. J. K., male; admitted March 28, 1905; age, eleven years. Lungs normal. Status with pulmonary

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edema every six to eight weeks. Occasional isolated grand mal seizures.

August 3, 1906. Temperature 103 degrees F. Seizures stopped on administration of chloroform, morphine, and atropine. Edema cleared up in twenty-four hours.

November 25, 1906. Status controlled by chloroform. Pulmonary edema and marked bronchorrhœa, with quantities of mucus from mouth and nose. Under treatment cleared up in twenty-four hours.

Case XI. C. H., female; admitted April 30, 1896; age, twenty-one years. Grand mal. Onset at fourteen years. No heart lesion.

March 7, 1905. This patient had several severe seizures last night and some today. This morning she was comatose and remained so all day. In the afternoon she had a slight elevation of temperature; pulse became very rapid, as did also respiration. Breathing was noisy, and laryngeal rales were present. Auscultation showed many rales in both lungs as a beginning of edema of lungs. She was given atropine sulphate, grain one-sixtieth, and strychnine sulphate, grain one-twentieth, hypodermatically. Latter drug repeated at intervals of two hours for three doses, then grain one-fortieth given every two hours.

March 8, 1905. Condition was improved. Very few rales heard over either lung. Patient coughed occasionally, but did not expectorate. She took nourishment by mouth, and was much brighter mentally. She did not answer when spoken to. When smiling, muscles of right side of face contracted more than did those of left side. She had a few mild seizures, which consisted of head being turned to left; slight lateral nystagmus and chewing movements; no automatism or stertor; duration about five seconds.

March 10, 1905. Patient has much improved, but did

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not talk. She took nourishment easily. Lungs were free from rales. Still some elevation of temperature.

August 10, 1907. Patient had serial attacks yesterday morning at 5:30. In spite of the administration of chloral and bromide in large doses, and thorough flushing of the bowels, the seizures continued, and pulmonary edema supervened. In the past twenty-four hours the patient had had forty-one seizures, and was in the morning at 8:00 in a critical condition; temperature 100.6 degrees, pulse 152, respiration 48. The edema of the lungs was increased in severity and the seizures recurred about every half hour. Nitroglycerine, grain one one-hundredth, administered at 8:45 a. m.; copious enemata of warm water; dry cups. Since last note her condition had become progressively worse. She had ten severe attacks between 5:30 a. m. and 12:55 p. m., and died at 1:05 p. m.

Autopsy: August 11, 1907. Froth about nostrils. Hypostasis fairly well marked and patchy in character present on upper chest and neck. Brain, large and soft, very moist, and congestion marked. Microscopical examination was negative. Both lungs showed moderate congestion and œdema. Heart weighed 230 grammes; there was large chicken fat clot in pulmonary artery; right heart contained mixed clot. Examination of valve was negative; endocardium was pale.

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DR. BROWER:

Mr. Chairman and Members of the Society: A very important fact was stated by Dr. Munson this morning, that I have realized for many years; that is, that there are many kinds of epilepsy. A not uncommon form is the one Dr. Hodges described in his paper, for which I propose the name of the gastro-intestinal form. In these cases we have two clinical conditions, brain irritability and auto-intoxication. In my experience I have been led to the conclusion that the condition of the abdominal cavity is not sufficiently studied. To illustrate: a woman aged about twenty-one was brought to me by her physician. She was having one or more grand mal attacks every week and was taking about 120 grains of bromide potassium daily. On examination, I found an atonic stomach and an impacted colon. She was taken to St. Joseph's Hospital, the bromides stopped, lavage, colonic flushings and abdominal massage resorted to. The diet was carefully regulated and ten grain doses of chloral given at bedtime on account of insomnia. She was kept in bed for four weeks and during this time was given general faridization. The chloral gradually reduced to five grains and forty grains daily of bromide added. This treatment has been continued now for about six months without any recurrence of the convulsive seizures.

I agree with Dr. FitzSimmons in his postulate that alcohol is a most potent factor in development of epilepsy, directly as well as by inheritance, producing a want of inhibitory power in the brain cells.

I appreciate with Dr. DeJarnette the great value of the bromides in the treatment of epilepsy, and at the same time know that their injudicious use has done great injury to some cases.

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DR. J. S. DEJARNETTE, OF VIRGINIA:

I have seen many cases of epilepsy after having one or two convulsions suck froth from the mouth into the lungs. The mucous rales made in the trachea and larger bronchii are distributed all over the chest, and the sound cannot be distinguished from œdema of the lungs. These cases will appear to be on the point of death, and in a few hours be able to sit up and walk around if they do not die. My first case of this sort alarmed me greatly, and I telegraphed to the patient's brother in California to come at once as he was dying. When the brother got to the hospital the patient was up and walking about, and seemed to be as well as I was. I have tried these cases with medicine and without, and have tried bleeding, but will never try *it* again. Chloral has given me better results than any other remedy. Sometimes in convulsions the patient will strangle if he falls on his face to the floor, or on the bed-clothes. Therefore some one should be with the epileptic all the time. I remember the first case of epilepsy I ever saw at the Soldiers' Home in Richmond. I thought chloroform had to be given at once, and as it was the precursive form of epilepsy, in which he ran a short distance and then moved around, I had a great deal of trouble in giving him chloroform. Before I could get him in the least under the influence, he came out of the fit, much to my relief. Never give a prognosis as to what an epileptic will do.

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THE RELATION OF EYE DEFECTS TO EPILEPSY

By D. D. Willcox, Petersburg, Va.

Mr. President and Gentlemen:—

In presenting this paper I wish to state that the title as printed on the programme does not altogether convey the exact meaning of my remarks.

Being an enthusiastic oculist I originally intended to show that eye diseases and defects were largely responsible for many cases of epilepsy, but after examining the 136 cases which I am about to report I have come to the conclusion that my contribution is but small evidence in proving that eye defects cause epilepsy.

To be able for the better understanding of how eye strain is brought about, we will first see what is meant by the terms, Hypermetropia or far-sightedness, Myopia or near-sightedness and Astigmatism or an unequal curvature in one of the refractive media of the eyeball.

Hyperopia or far-sightedness is that condition in which the eye is too short from the front of the cornea to the retina—consequently images are focused behind the retina instead of upon it, causing a blurring and consequently a continual strain upon the focusing muscle in the eye in order to see objects distinctly.

Myopia is that condition in which the eye is too long from cornea to retina and objects focus in front instead of upon the retina.

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Astigmatism is that condition in which the curvature of the cornea is unequal in some of its meridians, and causes an object to have more than one focus and forms a blurred image upon the retina.

Each of the above conditions, when corrected by a lens, causes a cessation of eye-strain.

A report of the cases examined at the Central State Hospital at Petersburg, Va., shows the following conditions:

Normal	41 cases
Cataracts in both eyes	7 "
" " one eye	2 "
Hyperopia simple	6 "
" with astigmatism	14 "
Hyperopic astigmatism	23 "
Myopia, simple	2 "
Myopia with astigmatism	1 "
Compound astigmatism	1 "
Far-sightedness in one eye and near-sightedness in the other	2 "
Scars left on eye from old ulcerations, impairing the sight	5 "
Cases having one useless eye from injury	8 "
Cases born with one useless eye from birth	4 "

Now, Gentlemen, we find nearly thirty per cent of absolutely normal eyes. The cases of cataracts in both eyes, numbering seven, were the result of old age. The two cases of cataract in one eye only were the result of injury. The other cases, represented by the irregular refractive conditions of the eye, certainly showed enough error to produce an incalculable amount of eye strain in an individual who uses his eyes to any extent in reading or

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writing or for any continuous near work, but all of these cases were negroes, nine-tenths of whom did not know their alphabet, so I do not think—after a careful consideration of the cases—that refractive errors play a role of much importance in the production of epileptic attacks. I do think, however, that as soon as epilepsy manifests itself the patient's eyes should be examined and a properly prescribed glass should be worn even if the patient be not more than a year old, because I believe that epilepsy is purely a reflex disease, where it cannot be shown to be due to traumatism about the head.

It is our duty to exhaust every possible means to get at the cause, and if the eyesight be defective from lack of glasses it is our duty to find it out and correct it before the epileptic habit becomes established.

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SURGICAL INTERVENTION IN THE TREATMENT OF EPILEPSY

By Stuart McGuire, M. D., Richmond, Va.

My gratification at being asked to read a paper before this Association is mingled with embarrassment at being called on to discuss a subject with which I have had less experience and to which I have devoted less study than many of those in my audience. I have nothing new to present and all I can hope to do is to review the work of others, and to state the conclusions I reach from the standpoint of a general surgeon.

Epilepsy has no constant morbid anatomy; it is apparently due to causes widely differing in character, and its treatment up to this time has been unscientific and unsatisfactory. Until a real knowledge of its pathology and etiology is obtained, and until a proper classification of its different types is formulated, epilepsy should not be considered a disease entity, but rather a symptom complex, the result of not one but of many different conditions.

The victim of epileptic convulsions is not always incurable, and patients should have the benefit of dietetic, hygienic, medicinal and surgical treatment, even though the means employed be empirical. The treatment should be of the patient—not of epilepsy—an effort to remove the cause rather than to relieve the symptoms. Success can only be secured by a special study of each individual case.

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It is folly to attempt to make a differential diagnosis between epilepsy, epileptiform convulsions, grand mal and petit mal, with the hope of finding a cure. Success, if success be possible, will only come from painstaking, searching, conscientious and skillful study of the individual affected.

The opinion of the profession in regard to the value of operative intervention in the treatment of epilepsy has changed with almost every decade. At one time it was thought that surgery was destined to cure a large majority of patients, and later it was believed that it only gave temporary benefit in a few selected cases, and that the results obtained did not justify the risks entailed. Neither extreme is right. Surgical operations should only be performed to meet logical indications. Surgery is not a cure-all, but will unquestionably relieve some cases which cannot be cured by other measures.

Operations which have been performed for epilepsy, and which have been followed, according to the report of reliable men, by complete relief of convulsive seizures, vary widely in their nature. In some cases the rationale of the cure can be explained; in others it is merely a matter of conjecture. An operation which leaves an opening in the skull will cure some cases, while one which seals the skull securely will be followed by cure in others. The removal of old cicatrices, especially from the scalp, has been followed by cessation of paroxysms, while in other cases the seizures have continued even after excision of the motor cortex. Resection of the cervical sympathetics, advocated by Jonnesco, has been followed by a cure in many cases, although the operation has recently lost its popularity. Ligation of the longitudinal sinus following accidental rupture, was so clearly a cause of cure in one

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case that the surgeon deliberately and successfully performed the same operation on a second, (Delangeniere). Phlebotomy has been advocated and been followed by relief of seizures, (Houzel) and doubtless during the so-called status epilepticus, such a procedure would be logical. Appendicostomy has been advised by LaPlace in certain selected cases where coprastasis is a prominent factor. The injection of eucaine into the motor cortex has been practiced by White and others, with apparent benefit. Cures have followed the administration of an anaesthetic, the removal of necrosed bone, the correction of uterine displacements and the operations of oophorectomy, dilatation and curettage of the uterus, clitorectomy, trachelorrhaphy, orchidectomy, nerve stretching, nerve section, circumcision, cauterization, removal of polypi from the nose, wax from the ear, adenoids from the throat, and section of the muscles of the eye. Finally, trephining or osteoplastic resection of the skull for exploration and the removal of the supposed cause of the convulsions, or of a portion of the motor cortex of the brain, have cured a large number of cases.

A study of the results of operations upon epileptics shows, according to DaCosta, that only from four to five per cent can be cured by surgical measures. This is not as good as it ought to be, but compares favorably with the result of medical treatment, which, according to Spratling, is only successful in relieving from eight to ten per cent of cases, and that when the patients are treated early, carefully and kept under proper control.

In estimating the value of surgical statistics, it must be remembered that cases may be definitely cured and then seizures return from an entirely different cause, or indeed, the recurrence may be the effect of a mutilating cerebral

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operation. This is especially true if the patient is by heredity or nervous construction predisposed to epilepsy. Certainly no one can expect operative intervention to give immunity to such a malady. Again it must be borne in mind that an operation which apparently fails at the time may ultimately result in a complete cure. Bullard, Putnam and Codman report cases which did not show decided improvement until the lapse of months or even years. Finally, we must not forget that many cases which are not cured are markedly improved, and that operations which are technically a failure are often of great value to the patient.

As a matter of fact, the results of the past and the possibilities of the future justify an active renewal of surgical interest in epilepsy. Because all cases cannot be cured is no reason to neglect those cases that are amenable to operative treatment. Cancer of the breast does not yield a high percentage of permanent cures, yet no one questions the fact that it is a proper field for surgical endeavor.

During the past we have certainly blundered. In spite of the fact that everyone knows that epileptiform convulsions are not always due to brain disease, there seems to be an instinctive inclination on the part of physicians and surgeons the world over to begin the attempt to cure the affection by an attack with either drugs or a surgical operation upon the cerebral cortex. Trustworthy clinicians have demonstrated that convulsions may be due to affections involving other organs than the brain or spinal cord, and the relation of distant lesions to epilepsy must not be ignored. Brown-Sequard produced epilepsy in a guinea-pig by injuring the sciatic nerve, and there is on record a large number of analogous cases in man. Whether or not there is any such thing as reflex epilepsy, the fact remains

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that in certain cases the removal of some source of irritation will be followed by relief of symptoms. In most cases the source of irritation has been of traumatic origin and located in highly organized tissue, endowed with abundant sympathetic nerve supply. The same mistake of treating symptoms rather than the cause has only recently been recognized, and is not yet entirely corrected, in the management of disturbances of digestion. For a long time the stomach has been subjected to much harsh and ineffectual treatment. It is now known that many of its disorders are due to reflex irritation from the appendix, gall bladder, liver, heart, kidneys and other organs. Just as some cases of dyspepsia cannot be cured by treating the stomach, but must be relieved by the removal of a source of reflex irritation, so some cases of epilepsy cannot be cured by operations on the brain, but must be relieved by the correction of distant, and frequently unsuspected, causes of irritation.

In deciding, therefore, on the line of treatment most likely to relieve a victim of epilepsy, the conclusion must be reached only after a painstaking study of the individual patient. An accurate history of the sufferer's previous life, of his diseases and accidents, of his hereditary tendencies, and of the exact character of his convulsive seizures, must be secured. A comprehensive and detailed examination of every organ and function of his body should be made, and, if necessary, competent specialists called into consultation. Any abnormality which might prove a source of real irritation should be removed. If this does not relieve the epilepsy, it will at least improve the patient's general condition, and a cerebral operation can be done later.

Excluding intra-cranial conditions, the most frequent

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seats of pathologic conditions which cause epilepsy are the generative organs, the digestive organs, the organs of special sense, and the large peripheral nerves. I have, in my personal experience, noted twelve cases where there was immediate and permanent cessation of epileptiform convulsions after radical operations for disease of the uterus and adnexa. Spratling states that many cures have been effected at Sonyea by abdominal operations on women. Goodell, years ago, stated that in many women womb disease existed only in the brain, and it is not surprising that their cranial brain is often dominated by the "pelvic brain" of Byron Robinson.

Results of operations on the digestive organs are not so striking. I have had one cure of apparent epilepsy to follow the removal of an inflamed appendix. LaPlace reports three cures after appendicostomy for chronic disease of the bowel. Other writers record cures after operations for hemorrhoids, gall stones and other affections of the bile tract. Operations on the eye, ear and nose have all been occasionally followed by good results. Eye strains should always be corrected. Disease of the ear, if present, should be treated, and polypi or other growths in the nose or throat removed.

Finally, after all possible extra-cranial causes are removed, if the seizures continue, an operation on the brain itself may be indicated. In these cases lies the danger of indiscriminate, imperfect and illogical operations. Every cerebral operation for epilepsy is more or less exploratory, and can only properly be done by the removal of a large osteopathic flap. The old trephine for this purpose is an obsolete instrument, and the Stellwagen trephine or De-Vilbiss' bone forceps should be used rather than subject the patient to the traumatism inflicted by the chisel and

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mallet. If a local focus can be found it should be removed, as in twenty per cent of cases a permanent cure has resulted. If adhesions are found they should be separated, as Starr reports many cases relieved by this method for from one to three years.

In focal epilepsy, with or without skull injury, a cerebral operation is indicated. The dura should be opened and the brain examined by sight and touch over the area exposed. Tumors should be removed; cicatrices should be excised; cysts should be drained; and any obviously diseased area in the brain tissue should be extirpated. If there is special increase of intra-cranial pressure, the cause of which is not removable, decompression is indicated and may result in cure. If nothing obvious is found, and if the attacks have been distinctly focal in origin, it is justifiable to excise the motor center from which the discharge seems to originate. In doing this the Faradic test should always be used. Excision of the motor center, however, is useless in cases of general epilepsy, and, as has been shown by Mills in some cases where the Jacksonian symptoms are merely a part of general epilepsy.

When Jacksonian epilepsy has followed an injury in the motor region, the chances of cure are obviously better than when the sensory area has been the seat of injury. Dercum, however, has reported one case in which Keen effected a complete cure by removing a lesion of the frontal lobe.

Friederich reports eleven cases of idiopathic epilepsy, all in men from seventeen to thirty years of age, operated upon after a duration of the symptoms of from four to twenty-five years. The results were great benefit in all cases, practically amounting to a complete cure in three.

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He differs from Von Bergman in the belief that operations should not be performed in long-standing cases, and proves that many of even these may be strikingly benefited, if not cured, by operative treatment.

In summing up the results reported from the Boston Hospital by Bullard, Putnam and Codman, we find that in thirty-nine cases, fifty-one per cent were benefited and forty-nine per cent were not appreciably relieved. This is certainly encouraging, especially when it is considered that many of the cases were operated upon before the development of the modern technique of brain surgery.

The conclusions I reach from my recent study of epilepsy are:

1st. Epilepsy is a symptom, not a disease.

2nd. Treatment should be directed to the cause, not to the result.

3rd. Success depends on the individualization of the patient and a careful study of the various functions of the body.

4th. When symptoms are not clearly due to lesion or disease of the brain, the surgeon's first effort should be to remove all extra-cranial sources of irritation.

5th. If after this is done the seizures continue, an exploratory operation on the brain must be considered.

6th. In cases where there has been injury to the head or where the symptoms are focal, an operation on the brain is indicated.

7th. All cranial operations for epilepsy are exploratory —a large osteoplastic flap should be lifted and conscientious and skillful search made for the lesion.

8th. Gross intra-cranial lesions should be removed, excessive intra-cranial pressure relieved, and finally, if no lesion is discovered and excessive intra-cranial pressure

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does not exist, if the epilepsy is focal in origin, the entire motor cortex should be excised after being carefully isolated by means of faradization.

9th. Statistics of the result of surgical intervention in epilepsy contained in most books embrace illogical operations, imperfectly performed with obsolete instruments, before the development of modern technique, and do not represent the prospect which can now be offered to the patient.

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OPERATIVE PROCEDURE AS A THERAPEUTIC MEASURE IN THE CURE OF EPILEPSY: A PLEA FOR A MORE FREQUENT SURGERY

By Matthew Woods, M. D., Philadelphia

In this paper I use the word "surgery" in its abstract sense, not concretely, or connected with sensible objects or in its specific relation to definite things, as, the amputation of a limb, the incision of an abscess or removal of a tumor, but rather the impression made upon the mind, nervous system and emotions by the immaterial concommitants of the surgical act, as well as the act itself—the combination of hope, fear, expectancy, dread—the impressions and experiences connected with anaesthesia, the sinking thought of passing through it into annihilation and nothingness, and all the other feelings of depression and momentary exhilaration known to us who have had personal operative experience.

The effect of these things, these mostly emotional things, I believe to be the cause at times of recovery from epilepsy, and may thus be regarded therapeutically because they cure just as drugs cure other diseases.

My purpose, then, is to point out that at times it may be the conditions connected with surgery—shock when it occurs, impressions upon the nervous system, disturbances of vasomotor equilibrium, and the like,—rather than anything uniformly definite that surgery does that causes sus-

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pension of epileptic seizures, and that the recovery occasionally following operative procedure is not necessarily due to the tangible thing that has either been removed or corrected.

Some cases get well spontaneously, proceeding as it were from internal impulse, others after removal of the exciting cause, although this is not always the case. Certain persons apparently acquiring the epileptic habit, as has been said, as a consequence of traumatism, tumor or other neoplasm, the convulsive tendency remains in spite of everything. Cures also are effected by the combined influence of drugs, diet and tranquility, by inducing, by ligation, an interruption of nervous energy between the skin and nerve centres, in cases of peripheral irritation, by removing the exciting cause, etc., etc., yet there is still a large percentage of cures that cannot be accounted for except in the way indicated above.

It is probably because of these impressions upon the entire system that so often, after almost any operation, the convulsions are absent for several months.

Even in cases where operations done for other diseases are followed by incidental recovery from epilepsy, such recoveries cannot always be accounted for by the theory that some cause or source of reflex irritation had been cut out by the operation, as this would be like justification for the ploughing of an entire field for hidden treasure, not likely to be found.

The absence, as far as we know, of an exciting pathology, to be sure, makes anything but conjecture impossible, yet the above hypothesis would seem to explain many otherwise inexplicable restorations, and this is the reason for the faith that is in me, not about the cases where surgery is the *only* remedy, but in regard to the

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occasional efficacy of surgery per se, *so that in otherwise incorrigible cases, after drugs and hygiene under capable auspices had failed to produce satisfactory results* or the slightest prospect of recovery, I would resort to surgery in hope of possible deliverance. Formerly justifiable operations for the cure of epilepsy by most people were limited to such conditions as fractures of the skull, with depression, etc., lesions of the brain or nervous system, or where the character of the convulsion indicated the presence of irritation in some specific region; which sometimes seems to bear the same relation to the convulsions that the button bears to the bell—press the button, the bell rings at the other end. Now, I at least feel that an operation for almost anything may cause either improvement or recovery, and that an operation for the removal of an ingrowing toe nail may be as much of a benefit as one for the removal of a tumor from the brain. This opinion has not been arrived at rashly but as the result of observation and experience.

The following line of reasoning may sustain my theory: Epilepsy is an apyretic paroxysmal neurosis, its victims neurotics, hence often easily impressed. Neuroses constitute the only class of diseases caused and cured by emotional excitement, or psychically and the like, as witness epileptic and asthmatic attacks and paroxysms of dyspnoea, excited in neurotics and others by odors, sights, apprehensions; secretions of gastric juice by the blowing of a trumpet, involuntary micturition, relaxation of the sphincters of the bladder by the squeaking of a bagpipe; or again, witness the cures effected on the neurotic by the laying on of hands, the Royal touch, by visits to shrines, pilgrimages to sacred places, by Christian Science, mental healing cures effected on the tarantati by the tarantella

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dance;* or again, ecchymosis caused by fright acting on an anxious imagination. The communication of the stigmata, an ecclesiastical illustration said to be supernaturally impressed upon the body of certain people in imitation of the wounds upon the body of Jesus, such as the stigmata of St. Francis, or, again, the deaf hearing, the lame walking, the dumb speaking, the blind restored to sight occasionally even in our own matter-of-fact everyday practice in consequence of emotions and experiences made by conditions connected with certain acts.

These various illustrations of the phenomena of disease, accepted as realities, are so incapable of explanations founded upon visible pathology, that, in accepting these, we do not need to doubt the possibility of epilepsy, a disease belonging to the same type, once in a while being cured in the same way.

Dr. Wm. Carpenter, in his Mental Physiology, a book that ought to be in every physician's library, says that so many mysterious things occur within the little universe of man so impossible of logical elucidation, that the educated person is not in a position to refuse belief in anything.

There are so many cases of recovery following operations, for example, for the relief of cerebral tension, when there was no tension, and the amputation of such parts as we know now had no connection with the cause of the disease, that I know of no other explanation except the one I have adduced. The theory may be too startling for universal acceptance nevertheless it seems capable of proof. Take the following illustrations from my own case book:

Mrs. B., wife of a druggist, aet. forty-two, had been an epileptic for twenty years, convulsions for the year

*See "De la danse de Saint Guy du XIV au XVII Siecle," E. S. Albans, Paris, 1872, also the Author's Mimetic Diseases.

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preceding accident averaging six monthly. For three months had been taking bromide of potassium twenty grains, three times daily in four ounces of water, hydrocyanate of iron one grain, three times daily, with restricted diet. During absence of the cook, Mrs. B. had temporarily assumed her place at the range. Breakfast being delayed, her son entered the kitchen and found his mother in a convulsion, the first one in ten weeks, with her charred hand rubbing against the bars of a red hot grate. An amputation under ether was done, and until her death, eight years after, she was free from convulsions, although treatment with epileptic medicine after three weeks' interruption, lasted only for a month after the recovery from the operation.

Louise, aet. twelve years, an epileptic since her second year, with convulsions averaging for the past year, twelve monthly, had been under my care three months, during which time she had but one convulsion. Treatment was bromide of strontium, ten grains four times daily in four ounces of water. Infusion of digitalis, a teaspoonful three times daily on alternate days, with selected diet and protection from excitement.

After a bath on a hot summer day her nurse sat her on a slipper box in front of an open third story window, went into an adjoining room for fresh clothing, and when she returned the naked child had disappeared through the opening, falling with such force on a wooden fence as to break it.

An hour later I found her on a sofa in the parlor excited, contused, with a deep laceration on the outer surface of her left thigh about four inches long, which, after washing and being brought together with stitches, under ether, she was put to bed. There were no broken bones.

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Until the family left the city four years after, the child was free from convulsions, the accident leaving no sequelae except a scar.

Margaret C. aet. nine years, an unusually irritable epileptic since her second year, with an average of three fits monthly, convulsions slight, short, almost psychic, more like what the French call *Vertige Epileptique*, was but slightly benefited by a treatment of bromide of strontium and infusion of solanum carolinense and adapted diet, became frequently interrupted and finally suspended altogether. Two years after she returned, spells more frequent, but with a bunch of tuberculous glands in the right surgical triangle, extending up toward the ear. Three years after their extirpation under an anaesthetic she is still exempt from convulsion and is entirely normal. In this case no epileptic medicine was given after operation.

It is such cases as these, and others somewhat similar, that have convinced me of the *occasional* benefit of almost any kind of surgery in epilepsy.

The combined concomitants of the operation, its awe, impressiveness and solemnity, as well as the act itself, acting psychically, in temperaments or minds specially susceptible as in the historic cases mentioned in the beginning of my paper.

I think this interpretation may be applied also to many of the cures effected under the direction of Brown-Sequard, and that his explanation of the removal of peripheral irritation could not now be applied to many of them. The same might be said of the numerous cures claimed by the advocates of the once popular Corsican Remedy,—cauterization with a red hot iron behind the ears. This popular site of reflex agitation then, could hardly, like

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the residence of an exiled Prince, have been relegated since, by the medical powers that be, to some more remote location. Fashions may influence diagnosis, but not pathology.

I think the same analysis may be applied to the many cases reported by Baker Brown, M. R. C. S., indeed, this surgeon was so successful in recoveries from epilepsy that his Society, the Royal College of Surgeons, thought he was getting up a corner in cures, and disciplined him. Mr. Brown, being convinced by Brown-Sequard's "Lectures on the Physiology and Pathology of the Nervous System," that great mischief was caused to the system in general and the nervous system in particular, by such excitement, decided consequently that epilepsy was due to storm of the "pudic nerve," mechanically induced, or rather that branch of it supplying the clitoris. Subcutaneous division of the nerve not being sufficient to effect a cure, Mr. Brown subjected his deduction to a more heroic surgical test, by removing the clitoris altogether. His amputations were done with a pair of scissors, afterward plugging up the scar with lint. In a month the wound was healed, and great numbers of patients were restored to health completely "with all the vital powers," as he attested, "greatly strengthened." He reported many cures six and seven years after operation and still free from spasms. For a time he also had many followers, when fashion again changed the habitat of the disease, this time to its former exalted location in the interior of the calvarium. Since then, besides the cures celebre following removal of actual tumors, etc., from the brain, Dr. J. Wm. White in the "Annals of Surgery," in two articles entitled "The Supposed Curative Effect of Operation *per se*," reports "extraordinary results from operations which

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proved to have no justification in a discoverable pathological condition." He collected ninety cases, in which trephining and other operations were performed, no lesion found, nothing else done, and yet marked relief, as well as complete cure followed.

I give a few varied illustrations from Dr. White and others.

"Drs. Agnew and White record the case of a man 'greatly epileptic' who years before received blow on head. Trephining was done, nothing abnormal was found. Eighteen months after operation he was still without convulsions."

"J. H. Hamilton reports a man, aet. eighteen, fits from childhood, one or two daily, the right common carotid was tied, which diminished the fits in force and frequency. He had *no* fits after the second operation, which consisted of ligature of left carotid."

This article also records instances in which permanent recovery followed tracheotomy, incision of the scalp. Dislocation of a bone of the great toe, where amputation was followed by permanent recovery.

Cures have also followed lithotomy, nerve section, nerve and muscle stretching, removal of cicatrices.

"Dr. S. E. McKenly, "American Medical Gazette," has reported the case of a boy, aet. sixteen, who, after having been an epileptic for years, fell through a bridge and had one testicle crushed. It was removed, and at the last report, six years after operation, there had been no fits."

Wm. H. Carr has reported the case of a man, aet. twenty-four, who had had fits for seven years, averaging three weekly. The operation of tracheotomy was performed for another disease; no fits afterward.

In ten cases of castration for epilepsy, all were helped and four reported cured two years after operation.

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In twenty-four cases of removal of the superior cervical ganglion of the sympathetic nerve, six had remained well at the end of two years, ten were improved, and five remained in the original condition, two died soon after the operation.

Twelve cases of epilepsy are reported as cured by such operations as stretching of the sciatic nerve, cauterization of the larnyx, circumcision, application of setons to the back of the neck, tenotomy of external recti, burning of scalp, etc., etc.

All of these recoveries would appear capable of the explanation given in the beginning of this paper, and constitute, it would seem, a claim for a more frequent surgery, but a surgery, though, in which the epileptologist should co-operate with the surgeon.

Philadelphia, October 24, 1907.

DISCUSSIONS

DR. WHARTON SINKLER:

Mr. President, I think in the majority of cases the cures of epilepsy are of short duration. Another point is that we only hear of cases that have been successful under surgical treatment. In many cases, even where the operation was minor in character, the results have been bad.

DR. J. ALLISON HODGES:

Mr. Chairman, I wish to say that we have enjoyed this meeting very much. On the subject of the surgical treatment of epilepsy, I would like to say a few words. I agree to a certain extent with what Dr. Sinkler has said, with this correction—that in many cases that I have seen treated by operation for the cure of epilepsy, the cure has been brief, as a usual thing for from thirty days to two

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or three months. In a certain class of cases, however, surgical treatment seems to have a more beneficial effect than in others. When a patient has clot on the brain or something of that sort, surgery is often beneficial. Some years ago I was talking to a superintendent of a hospital for the insane. He was interested in what I had to say and offered to send me patients to experiment upon, if I may use the word. He sent me three patients—severe cases of Jacksonian epilepsy—and I operated upon them in the hospital in this city. The superintendent of the hospital is now dead but his assistant is still living, and living on two of the three cases his superintendent sent me and on one other case which he had under treatment at the same time at which the others were operated upon. In two of the cases, one was discharged immediately from the hospital, the other in two weeks. A little later we took two other epileptics and made osteoplastic flaps, to see some of the workings of the brain. While we did not get any working of the brain, incidentally we helped this difficulty even if we did paralyze one side of the patient's face. The whole subject is an interesting one. I am convinced more than ever that surgery is beneficial and after listening to Dr. McGuire I am more surgically inclined than ever.

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INJURIES TO EPILEPTICS

By Alden V. Cooper, M. D., Massachusetts Hospital for Epileptics, Palmer, Mass.

In the entire list of diseases there is none which brings more danger to the patient from injury than does epilepsy, and one of the aims in the care and treatment of epileptics should be to lessen this danger as much as possible and to protect them in every way; as by suitable attendance, specially constructed wards and the like.

Even with the best of care all manner of dangers occur, for the patient in the throes of a convulsion and deprived of consciousness and all instinct of self protection, is exposed to all sorts of injuries which range from the slightest to the even fatal.

It is among patients having grand mal attacks without aura that the most constant watching should be done, and it is in this class that accidents and traumatisms are the most numerous. In those cases where there is a sufficient aura, many of the dangers may be averted by instructing the patient to lie down until the feeling or sensation has passed away.

The most common parts of the body which are apt to be involved in injuries are those which are the most exposed, such as the scalp, face, ears, neck and extremities. In the hospital where there are any considerable number gathered together, it is a matter of daily occurrence to have two or three or even more cuts of the scalp to sew up.

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Some may require only a single stitch, others require many. Most of such cuts are incurred by the patients falling against the edges of chairs, doors, tables or settees. Sometimes the patient gets a severe cut simply by the impact of the head with the floor and the consequent tearing apart of the tissues, they having fallen with such force; this is seen very often in patients who have no aura, and whose attacks come on suddenly. Besides incised wounds of scalp region, there are also many bruises and contusions varying in degree and intensity. Some patients constantly fall forward striking upon some portion of the face or forehead, and in such cases contusion and bruises of the forehead and the region of the eye are numerous. Also, we have a great many swollen noses, lacerated lips, teeth knocked out and the like. Frequent dislocations and fractures of the bones of the face are encountered. The nose and jaws seem to be the most common seats of dislocation and fracture. In the records of our cases we have several of dislocated jaws, either of one side or both. We have had a few cases where the patient has fallen forward striking upon the tip of the chin, fracturing the lower mandible, breaking and loosening several teeth. These have to be most carefully treated, and the patient, if he is having many convulsions and apt to fall upon the same area each time, should be kept in bed at rest until recovery has taken place. The bridge of the nose is often broken and the bones displaced in varying degrees. In one of the most recent cases where the nose was injured, the patient fell forward, striking upon his mouth, pushing his nose upward and backward. In this instance the end of the nose was torn completely from the upper lip, leaving a lacerated wound. With all such cuts of the face and scalp, we should do our utmost to bring about correct

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apposition of the parts and secure primary union if possible, thus lessening the ugly appearance of the scar and disfigurement of looks. In some cases the patient has a tendency to fall sideways, thus bringing the region of the ear into danger, with perhaps consequent impairment of hearing through injuries. Bruises, contusions and cuts in this region are quite numerous and frequent.

In the region of the upper extremities we get many cuts and bruises with frequent dislocation and fracture. We have had several cases of dislocated shoulders, elbows and wrists. One of the chief dangers of dislocations is that they may become chronic by being repeated through the strong convulsive efforts of the patient in succeeding spells, or that at least a dislocation may occur every time an attack happens. This is very well shown by one particular case which we have here at the hospital at the present time.

Case I. E. C. Female. Age eighteen. During convulsion on May 24, 1905, patient dislocated her left shoulder anteriorly. Reduced under chloroform anaesthesia. July 9th. Patient again dislocated her left shoulder during convulsion. Reduced under anaesthesia. January 22, 1906. Every time that patient has a convulsion now left shoulder is dislocated. During the attack, patient throws left arm up and then the head of the humerus becomes displaced. Arm is held with elbow away from side of body, hand turned palm upward. Several different appliances have been used to prevent this, but with little success. Today a plaster of Paris cap was made for shoulder joint to see if it would be of any benefit. April 2, 1906. Plaster of Paris cap has proved quite useful but at times the shoulder is still dislocated. June 21, 1907. Patient today was examined thoroughly and an operation

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was advised for shortening of the capsular ligament to rectify the dislocation.

Note:—Operation not possible as parents refused.

Fractures of the upper extremity are likely to occur from patient falling with hand outstretched, or by hitting of elbow or twisting of arm beneath body during convulsion. This past year we had a case of fracture of the olecranon process of the ulna, where the patient had fallen striking his elbow sharply upon the floor. In a couple of cases the clavicle has been broken by the patient falling forward and striking it against chair or settee. In one of the cases which happened here the patient while sitting in a chair had a petit mal attack, fell over backward and, hitting upon the outstretched hand, incurred a Colles fracture.

The lower extremities are not quite so commonly involved in serious injuries as the upper extremities, yet even here we may have serious trouble, as was illustrated in one of our patients who fell a short time ago, striking heavily upon his right hip. Upon examination it was found that the cavity of the acetabulum had been fractured. In the region of the thigh bruises and contusions are of frequent occurrence. Very often the shin bone gets bruised by hitting against edge of iron bed frames during the night when the patient has convulsions and disarranges the bedding. In this manner the patients here have become entangled in the bedding and as a result some of the nails of the toes have been torn away.

In some cases where patient is of hysterical type they may have an attack which will throw them to the floor and then, by the strong muscular contraction of succeeding ones, they may bound around over the floor hitting against chairs, settees, tables, incurring all kinds of minor bruises and cuts.

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In only one case here at the hospital has there been a fatal injury. In this the patient had a seizure of the procursive type, jumped out of a second story window, striking on the back of his head and neck, fracturing the fourth dorsal vertebra. This resulted in a complete motor and sensory paralysis below the seat of the lesion, and death resulted in about thirty-six hours from an ascending myelitis.

The general treatment for all such wounds, bruises, cuts and the like, should be the same as for any other surgical wound, but extra care should be taken that the dressings should be put on carefully and securely, since during convulsions they are often loosened and disarranged or even removed entirely. On account of this, good results are hard to obtain in the treatment of fractures in such patients, as by the frequent displacement of the parts and the bandages healing is retarded and the tendency towards deformity much increased. Hence such cases should be most carefully attended to.

In some cases where a patient has been injured during a convulsion, there seems to be a tendency for that person to fall upon the injured area during each succeeding convulsion. Why this should be cannot be said, unless it is due to the extra stimulation of the nerves in this area due to the irritation of the injury. We have had several of such cases here at the hospital, of which I will cite three of the most important. In these cases the wounds become chronic in character, will not heal, and become infected; much scar tissue develops, and a most detrimental effect is exerted upon the patient, lessening his general vitality and making his convulsions more frequent.

In such cases as soon as we observe the habit of falling upon the injured area, the patient should be kept at rest

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and constantly observed until the wound has healed, removing all source of irritation, for with the repair of the injury the patient once more seems to regain his normal condition and has less tendency to fall upon this area.

Case II. G. O. Male. February 8, 1907. During convulsion this morning fell on the ward floor, inflicting a severe cut over the right eye. Cut lacerated, about four inches in height, penetrating to bone. Necessary to take several stitches in wound to close it properly after it had been cleaned. February 14th. Wound healing nicely. No signs of inflammation have arisen. March 12th. Patient fell, cutting right eyebrow open again. Cleaned and dressed as before. March 18th. During convulsion dressing of right eyebrow disarranged and wound inflicted. April 21st. Eyebrow at present in good condition and healing rapidly progressing. April 27th. Eyebrow split open again. During every attack the patient falls forward striking upon this area. Very hard to approximate and dress properly this time on account of so much scar tissue being present. June 17th. Wound entirely healed some time ago. October 8th. Since April patient has not fallen so as to strike injured part. November 15th. While patient was up and working in ward had hard attack, falling face forward to floor, again splitting eyebrow open.

Case III. J. F. Male. February 14, 1906. Some months ago patient fell during grand mal attack inflicting a lacerated and contused wound over the left occipital region. Wound was antiseptically dressed and healed by primary union. Later, patient fell striking on same area, inflicting a more severe injury. Since that time whenever patient has a convulsion he falls striking on this same spot, so that in spite of treatment the wound has enlarged,

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developed considerable fibrous tissue beneath its edges, greatly retarding healing. Everything has been done to bring about its closure, but without success. Plaster of Paris cap was made for the skull and worn by the patient with some success. May 28. Permission being secured, head was operated upon for a radical cure. Edges of old wound loosened up and scalp in neighborhood of injury dissected back from skull. A sufficient amount of connective tissue was removed so that a good approximation of the edges of the wound was secured. Operation successful. July 2d. After operation was completed patient kept in bed and wound antiseptically dressed as needed. Head was in good condition and wound entirely closed.

Case IV. F. P. Male. At time of admission in 1905 patient had bruise on back of head due to its being struck during convulsion. This was made constantly worse by patient falling upon it so that finally there was a large chronic suppurating wound on the back of head. The patient's attacks were mainly grand mal with an aura, and each time the patient fell in a convulsion this area was injured. Everything possible was done; plaster caps were worn with some improvement, but healing did not take place. On October 23rd the head was operated upon. Wound was surgically cleaned and opened by free incision. On examination the scalp was found to have folded upon itself so that an outer portion of the scalp extended upward for about an inch and a half over the surface of the bone, thus preventing healing. All this was cleaned out and the surface of the skull laid bare. A great amount of old connective tissue was removed. After this was done the surface of the skull and the under surface of the scalp were curetted. The seat of the operation was made clean by washing with pure carbolic acid, followed by alcohol.

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Edges of wound were then drawn into apposition and closed with catgut sutures. No drainage was introduced, the idea being to have healing by primary union. Head was bandaged with considerable pressure over wound.

In a couple of days suppuration set in and it was found necessary to remove some of the stitches for drainage. The infection was gradually overcome by daily attention. At the present time the wound has entirely healed. In this case especially, the healing of the wound has brought about a great improvement in the patient's general health, for where formerly the patient was dull, stupid, careless of personal appearance, slow of speech and thought, he is now bright, neat, quick to respond to questions asked of him, and his physical condition has greatly improved. Now he rarely falls and strikes the head, whereas formerly it was an almost daily occurfence.

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MYOCLONUS EPILEPSY: REPORT OF TWO CASES

By E. A. Kennedy, M. D., Massachusetts Hospital for Epileptics, Palmer, Mass.

I wish to report two cases of myoclonus epilepsy occurring in male patients admitted to the Massachusetts Hospital for Epileptics.

The first case is that of a boy eighteen years of age, admitted to this hospital April 26, 1905.

The boy's maternal grandmother had some kind of a paralysis and died as a result shortly after. We are unable to find out just the nature of this paralysis. Patient's father was twenty-one years of age at the time of the birth of patient. He was a moderate drinker and died at the age of twenty-eight years of tuberculosis. Mother was eighteen years of age when she gave birth to this child, which is the oldest of her children. Mother has a very nervous disposition and states that she worried a great deal about trouble during her first pregnancy. Mother has suffered frequently all her life from dizzy and fainting spells. Patient has one sister and brother alive and well. One sister died at the age of one and one-half years of marasmus, and a brother died at the age of two and one-half of croup.

Patient was born at full term and weighed nine pounds. When a child suffered from intestinal indigestion at frequent intervals. He had measles at six months of age. At two years of age had a large abscess on left side of

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neck. Long linear scar at this point showing the seat of opening. While bathing one time got a very severe blow on head, the result from diving, and was confined to his bed one or two weeks.

Physical examination gives us a patient fairly well nourished with no well marked lines of deviations. Reflexes are all normal excepting the knee jerks which are slightly exaggerated.

Patient is a very hearty eater and complains frequently of slight distress after eating. He is suffering from a chronic gastritis of a mild form and is also very constipated.

He had his first grand mal attack at thirteen years of age and since then has had one to four hard convulsions each month.

His myoclonus paroxysms began when he was twelve years of age. He received a severe whipping at school and this was followed by a fibrillary twitching of the voluntary muscles, which he calls the "shakes." He was unable to control this condition and finally fell to the floor.

Since that time his paroxysmal attacks have averaged from two to ten and occurring at intervals from a few days to two weeks. These myoclonic attacks of epilepsy have occurred especially in the morning, when the patient first leaves his bed.

Should he be standing, the quick contraction of the muscles that flex the thighs upon the trunk throws him forward onto his face and hands with great force. He states he falls forward as if in the act of diving. Again, the contraction of some of the same muscles causes him suddenly to go to a sitting posture and then fall over on his back. If he should be sitting down he may be suddenly thrown forward onto his face and hands, or he may

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not leave the chair but severely flex the trunk upon the lower limbs. He rarely falls to the side.

As a rule, as soon as he reaches the floor he jumps up again and is none the worse for the experience. He frequently strikes his head during these attacks and is quite stupid for a while following.

He also has a general myoclonus. These attacks always occur before a grand mal convulsion and there is a general convulsive movement of the voluntary muscles.

Before he has myoclonic attacks he has a sudden attack of dizziness during which, he states, that the blood rushes to his head, he hears noises as if blood was working through his ears and the latter feel warm. He also feels sick around his heart and he states that his heart palpitates. Following the attack he feels well except for a frontal headache and some uneasiness around his heart.

The second case is a male twenty-three years of age, admitted to this hospital on November 23, 1902.

Paternal and maternal grandparents were able-bodied people and lived until well advanced years. Patient's father is dead, cause unknown. Mother is alive and enjoys fairly good health. She has a very nervous tendency and worried a great deal during the time she was pregnant with this child. We do not get any important history bearing upon this particular case.

Patient was a healthy child during infancy and was bright until his mind began to be affected as a result of his epilepsy. He had some of the children's diseases, but we are unable to find out just which ones. Suffered a very severe attack of la grippe at eight years of age.

On physical examination the patient is found to have a few stigmata of degeneration. Parietal eminences are found to be symmetrically prominent, the left being slightly higher

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than the right. Occipital protuberance is prominent. The lobules of the ears are adherent and he also has an enlarged nose. Patient has a high-arched palate and is always complaining of his heart being irritable. His heart sounds are normal. He has a stuttering speech and talks with great difficulty.

He began to have myoclonus attacks of epilepsy soon after he had la grippe at eight years of age. The spasms first involved some of the muscles extending from the trunk to the head. They caused a bobbing sensation to the head as a result of the fibrillary twitching. At times his head would be suddenly flexed upon his chest.

The attacks have become more general and affect all the voluntary muscles, and he has what he calls "shaking spells." If in bed he very often is thrown out onto the floor by the quick contraction of some of the strong flexors of the body. At times the myoclonus condition affects his left arm and shoulder only, throwing them suddenly forward. If touched at this time it will invariably cause him to have a more general myoclonus condition.

Both of these patients are well marked examples of myoclonus epilepsy and have been under close observation ever since admission to the hospital.

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THE HEART'S ACTION PRECEDING AN EPILEPTIC SEIZURE*

*From the Laboratory of the Craig Colony for Epileptics
By J. F. Munson, M. D., Sonyea, N. Y.

The etiology of epilepsy has been a subject of comment and investigation from the most ancient times to the present day, yet but little light has been shed on its mysteries. The recognition of the cortical origin of the convulsion, and, more important than this, the new view-point being adopted by epileptologists, which regards the convulsion or other paroxysmal manifestation of the disease as the symptom and not as the disease itself—these have been the most important steps in advance.

Out of the recognition of the symptomatic character of the attack has grown the idea that the disease is not a unit —is not due to the same cause confined to the same part of the body in every case. Instead, it may be said that every case is an epilepsy by itself; certain it is that there will be found to be several epilepsies.

In regarding the disease as a group of conditions, and not as a unit, we have to meet this question: How do different causes acting on the same cells bring forth the same action? Do they act directly or do they act indirectly through some condition produced by all in common?

Dr. Albert E. Russell, of London, has discussed this question[1] and answered it by suggesting cerebral anemia

1. Transactions of the National Association for the Study of Epilepsy, vol. iv.

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as a common condition produced by all the underlying causes of the seizure. Dr. Russell suggests that arrest of the heart's action may in some cases be the immediate cause of the anemia, the arrest taking place just before the onset of the convulsion, *i. e.*, before consciousness is lost and muscular contraction sets in, the failure to pump blood producing the anemia of the brain. In support of this assumption he cites cases, and I may be permitted to briefly summarize the material he presents:

He first quotes two cases (p. 42) with slow pulse, in which convulsions were preceded by cessation of the heart's action. His third case is one in which subacute rheumatism had set in. Benham, the attending physician, was informed one morning that the patient had had several fits, and while taking the pulse it stopped and a convulsion set in. This was repeatedly observed. The urine after these seizures contained albumin, but this soon subsided. The fourth case, in which myocardial disease seemed absent, was reported by Webster. Cessation of the pulse, shown by sphygmographic tracings, was accompanied, according to duration, by sleep or convulsion.

Russell next quotes a case of cardiac dropsy, and one of uremia (both Moxon's), in which severe epileptic convulsions were preceded by stopping of the heart's beats. A case of epilepsy, also Moxon's, in which cardiac arrest was observed, is mentioned. Dr. Hughlings-Jackson has observed the same thing in petit mal seizures. Dr. Russell reports one case from his own experience and gives the description of another which was reported to him. He concludes:

Sudden cardiac arrest is one of the few possible causes of anemia of the brain. It has been shown that such cardiac arrest does occur in some cases of epilepsy, and

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it is suggested that this occurence may be much more common than is generally assumed, and that the rarity of opportunities for making such observation may explain the fact that it is not more widely recognized.

This article and another[2] by the same author attracted my attention. The phenomenon observed and held to be the cause of the seizure is so simple and so easily observed that it seemed strange that it had not been noted before. Taking together the heart auræ present in some cases and the favorable action sometimes exhibited by digitalis in epilepsy, it did not seem improbable, especially when one considers the rarity of opportunities for observation, as indicated by Dr. Russel, that cardiac arrest might frequently be present.

THE AUTHOR'S METHOD

This suggestion, so simple and yet so important if susceptible of confirmation, has led to the present piece of work, which has consisted of obtaining graphic records of the pulse of epileptics previous to the seizure. Some six months has been spent in the work which, requiring the observation of the beginning of the seizures, even where material is exceedingly plentiful, is of the greatest difficulty. The time of the next seizure can not be foretold in any case, even approximately, and in order to obtain records it has been necessary to observe cases continually for hours and days at a time, a process which is tedious and laborious in the extreme. Casting aside digital palpitation as a physical impossibility, the author arranged an air transmission apparatus whereby the pulsations in a constricted limb were transmitted to a tambour recording on blackened paper. A Janeway sphygmomano-

2. Lancet, July 21, 1906.

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meter cuff was applied to the arm of the patient and inflated to some suitable pressure, recorded by a mercurial thermometer. The pulse waves in the arm were transmitted through the cuff and heavy connecting tubing to an expansile pouch of rubber, inclosed tightly in a glass funnel, which is connected with a tambour. The kymograph used takes a blackened record about seven feet in length, so that a whole day's record may be traced on one band.

I regret very much that no time-recording device was available for the work. Nevertheless, I am sure after several months' experience with this particular kymograph that its motion is practically regular—sufficiently constant for the work of this paper. The cases observed were taken at random, except that a few were selected because of heart lesions. The patients were observed lying in bed, sometimes bolstered up with pillows in any attitude comfortable for them. In reporting the observations on the cases I will give a short history of each case and then describe the findings.

Case I.—*History.*—Male, aged twenty-three. Grandmother a paralytic. Onset of epilepsy at fourteen, without assigned cause. Physical examination negative. Two tracings were obtained in this case. There was a very slight acceleration preceding the attack. After the seizure the pulse was regular and still slightly accelerated. In the second of these tracings the seizure began some fifteen pulse waves before the first muscular contraction is recorded. In the first the period was some two or three pulse waves shorter.

Case II.—*History.*—Male, aged thirty-three, a printer by trade. Had been deaf since eight years of age. Onset of epilepsy at twenty-five. Assigned cause, careless

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habits of eating and sleeping. Denied the use of alcohol; used tobacco moderately. Palate and chest asymmetrical; heart negative; dermographia. HCl present but once out of several examinations. The seizure (grand mal) observed began without a cry; there was a drawing up of the face muscles and the head turned to the right. Knees were slightly flexed. Pupils were dilated at the end of the seizure. In the tracing from this case, an accidental movement interrupted the curve before the attack, but about eight occurred before the beginning of the seizure. After the onset there was apparently a momentary slowing, but this disappeared and the waves were still closer together as they approached the first muscular contraction. The rate after the seizure was about the same as before.

Case III.—No. 1871. Male, aged twenty-five. Onset of epilepsy at nine, following scarlet fever and Bright's disease. Heredity negative. Thorax negative; dermographia present; some asymmetry of facial markings and atrophy about the shoulder girdle, especially in the supraspinatus and infraspinatus muscles. Mental condition fairly good.

The record of a grand mal attack is shown in the accompanying illustration. The tetanic convulsion was of considerable duration, the eyes were turned up and to the right and the pupils were dilated. The clonus was short. The tracing shows slight acceleration after the muscular contraction is recorded, and during and after the convulsion the heart rate is still more increased.

Case IV.—No. 2107. Male, aged twenty-six. Heredity poor. Pneumonia at one year of age. Onset at one year. Patient has had muscular rheumatism and has a presystolic murmur. Pulse regular.

Two records were taken—one of a grand mal attack,

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and one of a petit mal. In the first there appeared to be acceleration of the pulse rate before the attack, and in the second there was no change.

Case V.—No. 1580. Male, aged thirteen. Onset at fourteen months. Physical examination negative. In several tracings of petit mal attacks no change of rate preceded the seizure.

Case VI.—No. 1779. Male, aged thirty-one. Heredity poor. Onset at nineteen. Mitral regurgitation. Records were taken of four seizures, petit mal or mild grand mal in character. In one, there was no change in rate, but in others there was acceleration just before or just after the onset. The indicated onset preceded the first muscular contraction.

Case VII.—No. 846. Male, aged twenty-two. Mother suffered from severe headaches. Onset at ten, supposed cause a fall five years previously. In the tracings of two grand mal attacks, the first showed slight acceleration before the convulsion begins, and the second showed no change.

Case VIII.—No. 939. Male, aged eighteen. Onset at eight years of age. Heredity poor. Worm fits at two years. In this case I have only the record of a petit mal attack. There was only a slight acceleration.

Case IX.—No. 1004. A Jacksonian case, with almost constant clonic movements of the affected side. The tracings showed intervals in the convulsion during which the pulse was regular up to the point where resumption of convulsion hid the pulse wave in the tracing.

Case X.—No. 1524. Marked mitral insufficiency. Numerous mild attacks were traced in which the pulse was regular up to the moment of convulsion. Two other tracings showed no change in rate before the convulsion.

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These cases, chosen at random or for a short heart lesion, may be considered to represent the average of epileptics. Looking over the results of the work reported, it will be seen that the condition common to all but one was an acceleration of the pulse preceding the seizure and lasting through the onset stage to the moment of the first recorded convulsion. This acceleration was never great. The absence of a time-marker prevented exact statements, but the increases seemed to be, in the several cases, from eight to nine, or from twelve to fifteen or seventeen beats. In all the cases the pulse was entirely regular.

For this group of cases one can conclude definitely:

First—Cardiac arrest does not occur.

Second—Except for slight acceleration, and in one case slight slowing, the heart does not take part in the convulsion.

While I am certain as to the accuracy of the above stated conclusions, the question as to the extent to which they apply to all epilepsies is a different matter. It seems to me, however, that it is doubtful if cardiac arrest will be found in cortical epilepsies uncomplicated by heart lesions.

DR. WILLCOX:

In handling some epileptics who had been examined ophthalmologically one of them had a seizure and I had the opportunity to observe what was going on in his eye. Before the seizure, the arteries and veins were small, but about fifteen minutes after the attack, while coming out of his comatose condition, the veins were being dilated.

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SPHYGMOGRAPHIC TRACING DURING SEIZURE, SHOWING NO CESSATION OF HEART ACTION, REFERRED TO IN PRECEDING ARTICLE BY DR. J. F MUNSON.

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VOTE OF THANKS

At the session held on the evening of October 25th, 1907, the following motion was carried by unanimous vote:

MOVED: That the Association tenders a vote of thanks to the Governor of the State, the Mayor of Richmond, the City Council, and the Medical Profession of the City of Richmond and of the State of Virginia for the many courtesies extended to the Association during its sojourn in the city.

J. F. MUNSON, Secretary.

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OBSERVATIONS ON THE HISTOPATHOLOGY OF EPILEPSY

WITH TEN PLATES

"Post Hoc"*

"*POST HOC"—The Committee on The Prize Essay reported that no prize should be given in 1907, but the Committee recommended that honorable mention be made of the essay the motto of which was "Post Hoc."

This essay was submitted by Dr. E. E. Southard, 240 Longwood Ave., Boston, Mass.

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OBSERVATIONS ON THE HISTOPATHOLOGY OF EPILEPSY, WITH TEN PLATES

The present paper undertakes to describe the histology of certain cases of epilepsy from a functional point of view. The essential novelty of this description consists in the emphasis laid upon certain mechanical characters of the central nervous tissue in disease. The resulting theory might perhaps be termed briefly a microphysical theory, in a sense that it seeks a logical basis for the epileptical discharge in certain intimate alterations of pressure in the central tissues. But the term microphysical, besides being a little pretentious in view of our dense ignorance of the nature of nerve impulse, is also too narrow in view of further interpretation. That is to say, however convincing the data in the group chosen—late epilepsies—may appear, the structural considerations which develop are analagous and not identical with conditions in epilepsy at large.

The present study, whether it be termed microphysical, cytological, or what not, lays the emphasis no longer upon characters of nerve fibres or of nerve cells, or perhaps of their adnexa. The essay would rather call attention to certain pericellular conditions, giving rise to intimate alterations of pressure. At the present time we can merely describe these conditions and leave their translation into terms of volume energy and of surface energy to the future.

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The endeavor to describe pericellular conditions along these lines seems to be in a direct logical line with modern physiological work. No one has brought out more clearly than Sherrington the importance of what happens at the confines of the neurones, the *synapses*,[1] or (perhaps still more happily) the interneuronal *surfaces of separation*.

At these interneuronal or, more broadly, intercellular surfaces of separation, a wealth of reaction is possible. A somewhat formidable list of their possible activities is offered by Sherrington. Thus, they "might restrain diffusion, bank up osmotic pressure, restrict the movement of ions, accumulate electric changes, support a double electric layer, alter in shape and surface-tension with changes in difference of potential, alter in difference of potential with changes in surface-tension or in shape, or intervene as a membrane between dilute solutions of electrolytes of different concentration or colloidal suspensions with different sign of charge."[2] All this is perhaps as unknown as it is tantalizing; but it cannot be denied that histology can play some part in describing alterations which affect these crucial surfaces of separation, and so lay a foundation for future physical interpretation. Therefore, although modern interest in the synapse or physical surface of separation between neurones has been a physiological interest, the histologist must certainly enter the field, and define carefully what are the types of structural alteration in pericellular and intercellular regions.

Before describing the cases which underlie this study, let us briefly review, in the light of the considerations sketched above, some of the dominant hypotheses in the field of epilepsy.

Since the cases which form the immediate basis of this report are cases of late epilepsy, in which there is no

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proof of hereditary antecedents, it will be necessary to include in our review the general subject of organic epilepsy. Indeed, inasmuch as our argument hangs upon the possibility of parallel conditions in organic and in so-called essential epilepsy, it is important to bring out the part played by organic cases in the pathology of epilepsy.

The point of view of Hughlings Jackson has been chosen for the first consideration, both because these ideas underlie a somewhat large portion of modern work and because they are free from fashionable and perhaps perverse distinctions between the so-called structural and the so-called functional. After a review of the Jacksonian positions, a number of later contributions will be very briefly taken up.

The service of Hughlings Jackson to epilepsy consists, not merely in the definition of the cortical or partial epilepsy usually termed Jacksonian, but also in the suggestion of general principles of use in the definition of genuine epilepsy. There remains much that is physiologically sound in the *three-level theory* of Hughlings Jackson as applied to the nervous system at large; and the paroxysmal *discharge in the highest level* amongst nerve centers as the physiological basis of genuine epilepsy, must stand as an important suggestion for the pathogenesis of epilepsy.

The ideas of Hughlings Jackson, as particularly applied to epilepsy, have been given expression by a writer in Tuke's Dictionary of Psychological Medicine.[3] Anderson here maintains that there may be an epilepsy taking rise in the lowest nervous level, i. e. the spinal system together with its upward prolongation as far as the oculomotor nuclei. This *lowest level epilepsy* is exemplified in the respiratory or "inward" fits of children. The spasmodic

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bulbar disease of children, laryngismus stridulus, belongs to the same group. Anderson is inclined to include spasmodic asthma in the adult as an affection of this group showing bulbar instability.

The *middle level* of Hughlings Jackson is concerned with more frequent and definite epileptic phenomena; those, namely, of the partial type named Jacksonian. The limits of the middle level have never been laid down with exactitude. The middle level includes the corpora striata and the motor area of the cerebral cortex, as well as much of the sensory cortex, but excludes at any rate the prefrontal and occipital parts of the cerebral cortex. Paroxysmal discharging lesions in the middle level produce *epileptiform seizures* which, though they may be accompanied by unconsciousness, have no essential relation to unconsciousness and never begin with it. These seizures are associated with gross cortical lesions, and begin in the midst of clear consciousness with a localized spasm and, spreading in quite definite fashion, lead eventually to unconsciousness. In Jacksonian epilepsy loss of consciousness is an incident merely.

Not so in the case of genuine epilepsy, which is a matter of the highest nervous level and exhibits unconsciousness as the dominant feature. The highest level is situated in the anterior or prefrontal and posterior or occipital regions. These centers form a sensorimotor mechanism and are also the organ of mind—a sensorimotor mechanism, in the sense that they represent movements and impressions in complex combinations, and the organ of mind, in that consciousness arises and declines in connection therewith. The initial unconsciousness of genuine epilepsy is a sign that a discharge has taken place in the highest nervous level. The sequent universal convulsions

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signify a spread downward of the discharge to lower levels. Even the bulbar part of the lowest nervous level is likely to be affected for a time, but this part of the mechanism recovers first.

As a matter of course, the three-level theory of Hughlings Jackson can no longer be accepted bodily. Nevertheless, however much we shift the boundaries of our levels, we must admit that epilepsy as a high-level disease presents many attractions in theory. Moreover, one point stands out clearly as a point to settle. What, after all, is a discharging lesion? Wherein does a discharge in epilepsy differ from a neural discharge of the usual type? These questions appear logically prior to the question, Where do discharging lesions occur?

In the following paragraphs brief mention is made of various contributions to the pathology of epilepsy as bearing on the mechanism of discharge. No regard has been paid to the chronology of the researches cited, nor is any effort at completeness made.

The Jacksonian three level classification of epilepsies has already suggested how various in site of origin convulsions of this general class may be. From this standpoint the unity of epilepsy may well be called in question. The topographical method of research, successful as it has been in making out the general principles of neurology, has signally failed for epilepsy.

From a topographical point of view we have to consider (1) the body and the fluids at large, (2) the muscles, (3) the peripheral nervous apparatus, (4) the bulbospinal apparatus, (5) superior nerve centers.

1. The body at large and its fluids have been considered from time to time as yielding poisons of secretory or excretory character which give rise to convulsions. Peri-

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odicity in production or continuous formation with explosions after accumulation for various periods have been supposed. Examples of *non-nervous* conditions of alleged importance in epilepsy are the *persistent thymus and status lymphaticus* of Ohlmacher,[4] the *hypoplasia of aorta and great vessels* of various writers, autointoxication or perverted renal secretion (*paroxysmal hypertoxicity* of Voisin and Peron[5]), the *uric acidosis* of Haig, the *carbamic acidosis* of Krainsky,[6] certain *serum properties* of Ceni,[7] and infection of fluids bathing the central nervous system (*neurococcus* of Bra[8].) Without attempting to appraise the very various values of the above suggestions, we may remark that no one of them yields insight into the method of production of an epileptic convulsion. Experimental work with injections of blood and urine, uric acid, carbamic acid, *neurococci*, has so far yielded little or no information with respect to the mode of production of convulsions. Nevertheless it is conceivable that histopathological work upon the differential lines indicated in the present essay might yield results, despite the barrenness of the usual pharmacological procedures. The same suggestion obtains for work with the simple convulsions produced by alcohol and absinthe, or incidentally in uremia and diabetes.

2. The *muscles*, it would appear, must actually be normal or nearly normal in epilepsy (regarded as a disease showing repeated convulsions.) The fatty changes in voluntary and cardiac muscle, described by Mott[9] in the status epilepticus, can scarcely be thought to accompany each attack of convulsions. Mott's interesting finding is obscure, but may indicate the direction in which changes are proceeding in ordinary attacks of epilepsy.

It is within the range of possibility that a kind of epilepsy might be produced by a poison acting directly on

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muscular substance (for convulsions) and on central tissues (for unconsciousness.) It is possible that twitchings in the death agony are so produced. But the march of convulsions in ordinary epilepsy suggests a higher origin.

The case quoted by Gowers[10] to show the cerebral origin of epilepsy (case of Oebeke[11]) might be again quoted in this direction. The epileptic in question suffered apoplexy and the muscles of the paralyzed side never again shared in the hitherto generalized convulsions.

3. The peripheral and sympathetic nervous system fails to offer much light on the mechanism of epileptic discharges. Echeverria's suggestion concerning the sympathetic ganglia seems without foundation. The existence of epileptogenous zones in certain cases (compare Hughlings Jackson's case of a "boy who had fits when his head was touched"[12]) and in Brown-Sequard's experimental guinea-pigs[13] (trigeminal distribution,) not to mention certain characteristic auræ in epilepsy, suggests that the peripheral nervous system may be profoundly concerned in the initiation of certain fits. The "lowest level" fits of Hughlings Jackson and the "reflex epilepsies" of various writers render it difficult to exclude the thought that various irritations (peripheral or sympathetic) may give rise to convulsions, provided that normal checks and balances be removed from the discharging apparatus. It seems difficult to conceive, however, any possible differentiation of stimulus which could effect through the linkage of several neurones the characteristic convulsions of epilepsy. In any case we are left with no more insight into repetitive convulsions than what we possess for, e. g., strychnine convulsions. A consideration of peripheral nervous conditions leads us to the conclusion that some higher or central provision is necessary for maintaining

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the motor effects of a given stimulus beyond the normal period and with abnormal force. The inhibition of counter currents to a given motor discharge must evidently permit the continuance of that discharge until the exhaustion of the discharging cells.

4. Proceeding centralwards, the epileptologist has to consider the *spinal-trigeminal* type of epilepsy as produced experimentally in guinea-pigs and as observed to occur spontaneously in the offspring of the injured animals by Brown-Sequard. The great variation in the site of the effective spinal injuries suggests that the injury may produce or liberate a substance which passes through the cerebro-spinal fluid or blood of the guinea-pig to remote parts of the nervous system where the removal of inhibitions may be brought about. The occurrence of repetitive convulsions in the offspring without individual injuries supports this idea, since the chemical products of maternal injury may readily pass through the placenta. The conception of such placental passage is easier to manage than the conception of direct inheritance of injured spinal cords.

A consideration of Brown-Sequard's work consequently shows an epilepsy compounded possibly of two factors—(a) removal of inhibitions in some part of the nervous system by means of a product of the destruction of nerve tissue, and (b) an effective stimulus (trigeminal irritation).

The medullary epileptic centres of Nothnagel[14] and Schroeder van der Kolk[15] suggest again a *place d'armes* for the removal of inhibitions. The conception of such a center is not beyond the range of reasonableness; but there are as yet no sufficient histological data for arriving at any notion of conditions in these supposed centers.

5. The higher nerve centers are the favorite seat of

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modern work on epilepsy. Some writers, as Gowers, regard epilepsy as patently a cerebral disease, though of obscure character. It is evident, however, from our review of Hughlings Jackson, that to term a disease cerebral merely is to neglect to specify whether we deal or not with the sensorimotor and psychical mechanism. The cerebral character of epilepsy, were it admitted, carries us a short way only in the analysis.

As a fact, the higher centers are certainly the loci of the most extensive and important inhibitions, so that the synaptic tissues of the cerebral cortex in particular are approached with a degree of hope. The satisfaction of this hope along anatomical lines has been considerable. The intimate effects of the gross lesions found—a task of histopathology—have been less easy to decipher. The well-worn and efficacious methods of classical neuropathology —of cerebral and spinal localization—are of less service in these directions, and of almost no service when it comes to the elucidation of non-destructive lesions.

The best effort of classical and localizing neuropathology has been spent on the possible relation of Meynert's sclerosis of the cornu ammonis to epilepsy. As an epileptogenous center the cornu ammonis ranks with Nothnagel's medullary center. The possibility, nay in some cases probability, of association between these centers and epilepsy is certain. The manner of this possible association is not clear. The usual form of critique is to point at cases without such centers and stringently require some unique and disparate center for satisfactory explanation. As a matter of fact the present superficial review indicates that we must probably accept what may be termed a manifold causation for epilepsy. If this is granted, we must evidently seek the intimate mechanism in type cases. The

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task is to show what may be the basis of the inhibitions necessary for the production of repeated convulsions.

An enumeration of the manifold anatomical causes which have been alleged in epilepsy is almost superfluous—foci of softening in central or temporal cortex, sclerosis of cornu ammonis, gliomata of various regions, dural endotheliomata, calvarial thickenings, chronic leptomeningitis, chronic encephalitis, focal lesions of a chronic inflammatory character (including tubercles and gummata.)

So far as those who report these lesions regard them as causative of epilepsy, in the sense that internal capsular hemorrhage is causative of hemiplegia, these modes of explanation seem destined to failure.

Another path is open. In common with several other workers, the present writer feels that the proper mode of explanation must be a cytological one. Conditions at the synapses must be investigated. The methods for studying nerve tracts should be discarded for the present purpose and replaced with finer histological methods.

The histopathologists have worked to some extent in the field of epilepsy. Their findings are usually taken as secondary. Thus Adolf Meyer,[16] in a highly critical review of the pathology of epilepsy, remarks: "Histological findings of the brain yield little that could be called causative. The occasional gliosis of the cortex discovered by Chaslin and Bleuler, and corroborated in some cases by Alzheimer, Weber, and others, and the nuclear changes shown by Bevan Lewis,[17] are not explicable, except as a secondary condition." It is evident that the changes described by Clark and Prout[18] and by Mott[19] are liable to the same charge.

Perhaps we should not throw out of court some of the most precious material for the elucidation of the general

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problem. The present writer is inclined to think that, by combining the consideration of *gliosis* on the one hand and of certain *stratigraphic changes* on the other, a structural basis can be laid for the understanding of the inhibitory mechanism which underlies epilepsy.

Before inquiring what these significant changes are, it may be well to forestall certain objections.

Thus it may be alleged that the alterations described below, being found in cases of late epilepsy, are not only of suspicious value for any special purpose, but also can obviously have no relation to the problem of idiopathic epilepsy. To the latter objection I may reply that, in the case of idiopathic epilepsy, we are often in the position of some one who had inherited the second generation of Brown Sequard's guinea-pigs without knowledge of injuries to the first. We are obviously better off if we examine cases of epilepsy whose histories we know, with respect to date, manner of origin, and course. Naturally the conditions of inherited epilepsy are not reproduced with exactitude in these cases; but surely, were we keen enough, we could make out the elements involved in the first instance. And, if it be interposed that the hypothesis of inherited toxines is pure speculation, I reply that, after all, I do not use the hypothesis in interpretation of specimens, and the neglect of cases of epilepsy, just because they show lesions, is an unphilosophical neglect.

But the former part of the objection—viz. that the changes, being found in admittedly organic cases, have little differential value for our purpose—is more serious. To this I would urge that the data of cortex histology are at the present writing not wholly obscure—witness the work of Ramon y Cajal or the work of Campbell.[20] That is to say, there is a certain outstanding stock of knowledge

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which permits us to say roughly where in the cerebral cortex and *in what layers* certain operations go on. As general topographical relations begin to be understood, there is becoming clear a certain movement in knowledge of stratigraphical character. And, although the doctrine of inhibition is still inchoate, we are not quite destitute of contributions thereto.

The cerebral cortex is evidently the most promising field for work, since it contains synapses of primary importance. The well-known cases (one of which was cited above) in which hemiplegia supervening in epilepsy suspends convulsions in the paralysed side, throw interest back upon the cortex. Moreover, Prus[21] has shown that painting the motor cortex with cocaine will prevent generalized convulsions, although it fails to suspend the electrical irritability of the cortex for single muscular movements. Now the single muscular movements are, almost beyond doubt, initiated by activity in the characteristic large cells of the precentral region (giant cells or cells of Betz.) When cocaine is locally applied to the cortex (or when bromides are administered,) we have to suppose either a general or else a differential diminution of irritability in the nerve cells of the cortex. In case this heightened threshold of stimulation is general, then we must suppose that electrical stimulation breaks through more speedily for the great motor cells (perhaps by reason of their relatively extensive receptive surfaces) than for the remainder of the cortex. Or in case the heightened threshold be differential, we may suppose the cocaine or bromide to leave the motor cells unaffected and to act upon the remainder of the synaptic apparatus.

Proceeding to an analysis of the functions of the motor cortex, we observe, of course, tremendous gaps in knowledge.

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In the first place, as taken for granted above, the giant cells must be regarded as analogous to the interior horn cells of the spinal cord and to the Purkinje cells of the cerebellum, namely, as the discharging elements for that portion of the apparatus to which they pertain. It is therefore by means of these cells that some kinds of epilepsy are rendered possible. The remainder of the motor cortex appears to have three possible functions (exclusive of unknown ones)—the *reception* of stimuli from elsewhere and the modification of motor discharges in one of two directions, either through *inhibition* or through *facilitation* of the discharges. As for the reception of stimuli, we do not stringently require the allotment of a separate type of cell: the receptive surfaces of the motor cells could readily take up energy directly from instreaming fibers (from the postcentral gyrus or elsewhere.) The functions of inhibition and facilitation seem to require more specialized apparatus. Most workers assume that these functions are performed by the outer layers (on account of various evolutionary considerations which do not here concern us.) Thus, according to this conception (which is not far from the removed current conception), the motor cells are those elements upon which the remainder of the cortical elements play, checking or hastening their impulses and perhaps modifying their force of impulse.

We considered above that the muscles must in the great majority of epilepsies be normal muscles. Just so with the motor elements and the intervening tracts: as a part of the transmissive apparatus, both spinomuscular neurones and corticospinal neurones must, to permit epilepsy, in the majority of cases be functionally normal. Just as it would be immensely difficult to procure conditions at the surfaces of muscles which would permit a simulation of

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the epileptic march of contractions from group to group and from limb to limb, so also it would be a complex circumstance which would permit the same event through effects wrought upon the surfaces of the anterior horn cells. Higher in the axis, such generalized disorder becomes more easy to produce, perhaps by reason of the intercalation of the cerebellum and of Monakow's tract.[22] The possibility of a cerebellar type of epilepsy cannot be denied even on clinical evidence. But such types and the bulbar or pontine types of various authors must represent a combination of circumstances greatly less frequent than those cortical conditions which admit epilepsy. And the central phenomena which underlie the epileptic march of convulsions—the "occasional, sudden, rapid, and excessive discharge" of Hughlings Jackson—must happen in these subcortical cases within so small a space as to be extremely difficult to unravel histologically.

We have analytically arrived once more at the cerebral cortex as the most promising field of work. Therein exist the motor cells as so many filters through which the nervous currents are poured. These filters are of a certain capacity, and they become occluded to nervous currents after a time (or so we may express the phenomenon of exhaustion.) What now governs the amount and character of the currents which reach the filtering surfaces?

We must be wary of speculation concerning the nature of nerve impulse, at least for the present purpose. What have epileptologists found in the cerebral cortex which might influence the amount and character of impulses to be let through the motor cells?

In the first place, there are certain vague ideas afloat concerning the possible epileptogenous character of variations in intracranial tension. Gowers, in the latter part

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of his second edition, speaks with a certain ridicule of various trephinings in epilepsy based on these vague ideas. Nevertheless, for fifty years since the carotid compressions of Kussmaul and Tenner[23], these ideas have had a certain justification. We are able to reconcile these findings in several ways with more special ideas, and, in case the Kussmaul-Tenner contractions are not of subcortical origin, we can still invoke the differential action of cerebral anaemia upon the various parts and layers of the cortex.

Secondly, epileptologists have busied themselves with local (intracortical) vascular conditions. A revival of conceptions of this group is seen in the latest book on epilepsy accessible to me.[24] Herein J. Turner describes epilepsy as "a disease occurring in persons with a defectively developed nervous system, indicated by certain structural peculiarities, and in whom there is a special tendency to intravascular clotting; the convulsions are a symptom of the disease, and prior to a seizure a condition of cortical stasis is induced by the formation of intravascular coagula." Whereas none will doubt that there are cases of epilepsy based upon intracortical thrombosis, it is probable that Turner's hypothesis is of too general a character to prove satisfactory in the end.

We now approach considerations at which it is less easy or desirable to blink, namely, a very general allegation of cortical gliosis in epileptics and the cell-findings of Bevan Lewis and of Clark and Prout.

With respect to gliosis, it was developed above that the condition is usually deemed secondary. The present writer feels that the significance of gliosis in epilepsy may be attacked on another line: the condition is altogether too generally found to permit its unequivocal acceptance as intimately related to epilepsy. Gliosis of a particular

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character and cocus, as will be developed below, may attain a separate and important meaning for epilepsy. The gliosis required is evidently not a gliosis following destruction or motor elements or a gliosis following generalized atrophy, but a gliosis having special relation to the non-discharging part of the motor cortex.

The writer therefore agrees with those writers who allege the frequent or even virtually universal finding of gliosis in cortical epilepsies, but is not satisfied that all of these glioses have been properly localized or interpreted. Especially is this true of the superficial cortical gliosis of many writers: this, the most frequent of significant lesions in the brains of adults, must be quite particularly investigated before accepted as epileptogenous.

The important stratigraphic contributions are those of Bevan Lewis and of Clark and Prout. The second cortical layer is the locus of lesion in both studies, and this unity of opinion is the more remarkable since the later writers (Clark and Prout) do not appear to have considered Bevan Lewis' work as extremely important.

Bevan Lewis[25] described certain cell changes, demonstrable by fresh methods, as characteristic of epilepsy, although found to some degree in alcoholic brain-disease. The characteristic lesion is a degeneration of the small nerve cells of the second cortical layer. The nucleus is first injured and presents in its center a refractile fatty body. Later stages of the injury may show the nucleus occupied by two such bodies, or completely replaced by one. At a stage when one-half of the nucleus is occupied by such a body, the nucleus begins to stain poorly and to be scarcely differentiated from the cell-body. The cells themselves persist for some time. The cells of other layers besides the second are occasionally affected but not

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universally. The fatty bodies eventually enlarge and burst, leaving the cells strikingly vacuolated. Along with the fatty nuclear change and vacuolation, Bevan Lewis says the "neuroglia is, as long as known, invariably in excess of the normal; but there is no increase of adventitial nuclei or of spider-cells."

Parallel with the above changes in idiopathic epilepsy, Bevan Lewis describes certain other findings as characteristic of epileptic idiocy, viz. inflated spheroidal cells with few processes in the same (second cortical) layer.

These stratigraphic lesions of Bevan Lewis suffer from the lack of sufficient indications whence the sections were derived. Moreover his induction that the sign of the convulsive constitution is a disparity between nucleus and cell-body, with alteration of the nucleus, is over-bold and probably too general.

Turning to Clark and Prout,[26] we find these writers incriminating the same second cortical layer. Curiously enough, these writers again allege characteristic nuclear changes. Most of their work appears to have been done with the motor cortex; but topographical description is for the most part lacking.

Chromatolysis was found by Clark and Prout in all types of cortical cell, most marked in the large pyramidal cells of the third layer. Some cell bodies stained diffusely. some showed finely granular chromatic substance. The type of chromatolysis varied. Vacuolation attended the chromatolysis.

The nuclei of the cells throughout showed changes, but particularly the nuclei of the small pyramidal cells of the second layer. These nuclei are swollen and granular and often without distinct membrane. In the cells of the third layer, the nuclei are often distorted, small, pale,

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or have "almost disappeared." The karyoplasmic network of the nucleus was often absent and replaced with a lightly staining granular mass. The nucleolus—and here lodges the most remarkable portion of Clark and Prout's findings—was found displaced to one side, especially in the cells of the second layer. The nucleoli were often abstracted in the line of direction of the knife employed in cutting the sections. More than sixty abstracted nucleoli were found in a section with a surface of one centimeter.

Disregarding certain details of description, the findings of Clark and Prout correspond with those of Bevan Lewis, in so far as they point to the outer portion of the cerebral cortex and particularly the second cortical layer as the essential locus of disease in epilepsy.

The present writer feels, as the above analysis shows, that the second cortical layer and the intermediate layers are certainly the loci to examine on theoretical grounds, since therein must lie the apparatus for controlling and modifying motor discharges.

In an experience of towards a hundred autopsied cases of epilepsy of many types, the writer has examined both the nervous and neuroglia structures with greater or less care. The phenomenon of gliosis in the subpial and perivascular regions is so constant as to be at first blush very promising. But this finding proves, if anything, too much; and indeed a sclerosis of greater or less degree is found in so many cases of nervous disease that are not epileptic that little can be induced from the finding with respect to epilepsy. The loss of a certain transmissive or associational function can probably be assumed from this superficial lesion. In passing it may be noted that the topographical analysis of lesions of this stratum, as

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evidenced by methods for neuroglia, might prove of greater service to mental disease than the more usual analysis by myelin sheath methods. As is well known the lips and basis of the gyri are more apt to show a dense gliosis than the crowns of the gyri. In epilepsy this finding—which may be significant of difficulty in transmission from gyrus to gyrus—is not particularly striking. On the contrary I have found rather more often a tendency to stringently focal gliosis, which are found upon the crowns of the gyri perhaps as often as at the bases. Plate I, Fig. 1, illustrates this finding and exhibits the cortex of the motor area (arm) overlaid with neuroglia fibrillary tissue in three distinct nodules separately by unaffected tissues. Such appearances can be found in numerous brains upon search; in certain cases of organic epilepsy, the finding is practically universal. The similar focal distribution of neuroglia tissue about a cortical vessel from the same case (Plate I, Fig. 2) shows how chary we must be of making functional inductions from findings of this character.

The writer is therefore skeptical concerning the significance of subpial gliosis for epilepsy, though seeing therein evidence of a transmissive disorder of great interest topographically in mental disease. This statement seems unconditionally true for the gliosis of uniform depth (or gradually augmenting at bases of convolutions) which is so often seen. And, despite the greater incidence of a more focal type of sclerosis in the writer's cases of organic epilepsy, he is wholly prepared to admit their dependence upon more general conditions than the epileptic discharge. Probably most workers would agree that such findings, if significant, are in any event secondary.

There are glioses of a different order of significance. In connection therewith the following case may be pre-

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sented. The case is anatomically extraordinary and affords some little color to the well-known cornu ammonis theory of epilepsy. The epilepsy had lasted only nine weeks (although there had been shaking attacks for a year or more) and was ended by a series of convulsions, amounting to status, lasting four days. The autopsy showed intense intracranial congestion with a deep red line in the midst of the right cornu ammonis. Plate II shows conditions in the cornu; injection, extreme overgrowth of neuroglia cells, from without inwards, *coupled with no demonstrable loss of nerve cells*, except perhaps in the outermost layer.

Before discussion, the details seem worthy of presentation:

CASE I*

The patient, a fisherman, born in 1836, had a number of insane, but, so far as could be learned, no epileptic relatives. Scarlet fever in childhood had left him slightly deaf. There were two accidents with unconsciousness, one a fall from mast-head at fifty-four or fifty-five years with unconsciousness three or four hours, the other at sixty-five, unconsciousness from vapor on a launch. A gradual decay of faculties began at sixty-six years, failure of memory, depressive ideas, train of thought limited to fishing, attacks of trembling without unconsciousness, irritableness. At sixty-eight years, nine weeks before death, the patient began to have fits which were at first severe but infrequent (three, once with a slight shock, in the first three weeks,) then grew less frequent but remained severe, and ended by four days of practically continuous convulsions with unconsciousness (over 400 attacks.) The convulsions were general, clonic, with head and eyes turned to left, right arm flexed over abdomen, thumb

* I am obliged to Prof. A. M. Barrett for the material of this case, drawn from his Danvers service.

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drawn under fingers, left hand extended by side. At times there were tonic convulsions.

The autopsy (four hours after death) showed the following conditions:

Intracranial congestion.
Focal encephalitis of right cornu ammonis.
Bronchopneumonia of hypostatic distribution.
Coronary arteriosclerosis with calcification.
Aortic and renal arteriosclerosis.
Chronic valvular endocarditis.
Chronic fibrous myocarditis.
Fatty myocarditis.
Chronic passive congestion of liver and kidneys.
Marked chronic external adhesive pachymeningitis.

Following is a description of findings in the head:

Calvarium not notable. Dura mater completely and firmly adherent to calvarum. Sinuses negative. Arachnoidal villi moderately developed. Pia mater dark in color, with veins intensely engorged. Pia mater a little hazy and in the frontal regions distended with liquid. None of the intracranial arteries show microscopic signs of arteriosclerosis. Weight of encephalon 1,270 grams. Blood content of cortex high. There are no notable anomalies in size or arrangement of gyri. The gyri are as firm as normal except that the cornu ammonis are a little firmer than usual. Section of the encephalon in various parts shows intense injection of the brain substance affecting both gray and white matter. The right cornu ammonis shows a grayish red linear streak running about the convolution in the nerve cell layers. This has an anteroposterior extent of perhaps 1 cm.

There is no need for the present purpose of analyzing finely the relationship of symptoms and findings in this case. Probably there exists in the literature no more demonstrative case of a virtually non-destructive lesion of the cornu ammonis which can be reasonably related to an epilepsy which was acute. A focus of irritation elisted in

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the right cornu ammonis which, whatever its cause, exhibited an interstitial accumulation of neuroglia cells leaving the nerve cells intact. It does not require undue stretching of the facts to allege that new and unusual surfaces of separation (to use Sherrington's phrase) had been provided for the nerve cells in question.

Suppose that these cells subserve some sensory function (as that of smell), there is little doubt that a continuous stream of impulses is impinging upon these cells, causing them to discharge along their normal lines. Their discharge may occur to the point of exhaustion, or, if not so violent and continuous, the discharges of the next neuronic links may be limited by exhaustion or by counterstreams or intercurrents from other regions. At all events, whatever the details of spread (and those may well be beyond the range of investigation,) the elements of a hypothesis stand out and get illustration from the above case: *Fresh surfaces of separation have been interposed between sets of nerve elements.* Those elements which have their currents in the forward direction, being placed under fresh conditions of intimate pressure, initiate continuous or lasting streams of impulse which set the remainder of the apparatus moving in abnormal fashion. Supposing, for the sake of illustration, that these particular intimately compressed cells formerly subserved the sense of smell, it might be inquired whether some unusual and violent olfactory stimulation at the sensory surface might not produce epilepsy. An affirmative reply is wholly permissible according to the present hypothesis; but the combination of circumstances which would permit such a result is simply unusual. The above case of focal encephalitis of the cornu ammonis may be therefore said to demonstrate clearly but one of many possible chains of events in epilepsy.

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Let us now examine a second case of epilepsy which presents somewhat similar features, but which ran a chronic course and falls more readily into the so-called organic group.

Our second case is also one of unilateral cornu ammonis lesion. The symptoms in this case can be traced to an aneurysm of the left anterior choroid artery which compressed both the uncus and the superior temporal gyrus of the left side. It is fortunate that the date of this aneurysm (or at least of a hemorrhage early in the aneurysm's development) can be assigned with considerable exactitude, because the patient was taken to a general hospital at the age of forty-four suffering from her original convulsions. Epileptic convulsions of mild character ensued, but there was no marked mental failure for ten years or more. The patient died a confirmed epileptic at the age of sixty.

Details of the case follow:

CASE II

M. Y., female, born in 1848. One sister insane. An aunt epileptic. Nine other sisters and brothers without history of insanity. Education poor. Mental capacity good. Married at eighteen years of age. Gave birth to five healthy children. At the age of forty-four, she was brought to a general hospital, having "fallen in a fit" and vomited in the street. Respiration of Cheyne-Stokes type. Pulse fair strength and volume, regular. Right external strabismus. Right pupil dilated and stationary to light. "Chewing motion of jaws." Before nightfall the next day she became rational and could answer questions tardily. Semi-consciousness was maintained without change of symptoms for twenty-four days, whereupon a sudden attack of nausea and vomiting with cyanosis and slight twitching of arms supervened. She grew gradually brighter during the next fortnight, but afterwards, about seven weeks after the original admission to hospital, began to complain of

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a numbness in the right hand. Her general condition gradually improved and she was discharged nine weeks after her accident much relieved.

After this attack the patient had frequent slight epileptic convulsions, affecting the right side. Her mental power gradually failed. Failure began to be particularly extensive after the age of fifty-five. The patient developed visual hallucinations at sixty years of age, and grew gradually worse until she became confined to bed eight days after admission to an insane hospital.

A somewhat unusual array of skin lesions was presented, suggesting decubitus in some ways, but far more extensive than usual. The left foot was greatly swollen and blistered. The pupils were equal. The right pupil was stationary to light. Sensation, as far as demonstrable, good. Knee jerks and Achilles reactions brisk, on both sides. Brisk Babinsky reaction on right side. Plantar reaction absent on left side. Elbow and forearm reflexes exaggerated. Abdominal reflexes normal. Organic reflexes impaired. General muscular weakness with incoordination and inability to stand or walk. The patient's utterances were confined to a few oaths.

The patient showed no convulsions while in the hospital and died two days after admission.*

The autopsy (seventeen hours after death) showed the following conditions:

Multiple ulcers of skin (decubitus ?).
Fatty change of heart muscle.
Chronic diffuse nephritis.
Slight aortic sclerosis.
Mitral and aortic valvular sclerosis.
Bilateral hypostatic pneumonia.
Chronic adhesive pleuritis of left apex.
Chronic splenitis.
Slight chronic external adhesive pachymeningitis.
Dilatation of lateral ventricles of brain.

* I am obliged to Dr. H. A. Cotton for these notes drawn from his Danvers service.

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Chronic internal hemorrhagic pachymeningitis of vertex in both hemispheres.

Flattening of convolutions.

Focal basilar arteriosclerosis.

Aneurysm of left anterior choroid artery, with atrophy of left uncus and compression of superior temporal gyrus.

Herniation of lips of superior temporal gyri.

Dilated perivascular spaces of left basal ganglia.

General encephalomalacia and myelomalacia.

Following is a description of findings in the head:

Hair red mixed with gray. Scalp more adherent than usual to calvarium. Calvarium contains a moderate amount of diploe. Dura mater slightly adherent to calvarium in vertex region, but more than normally adherent to base of skull, especially in middle fossae. The bone underlying this region of adhesion is slightly porous. The inner surface of the dura mater on both sides over the vertex and superior surface of the cerebrum is underlaid by a delicate transparent, slightly brownish membrane, measuring nowhere over 1 to 2 mm. in thickness, and playing out gradually over the flanks of the brain. Sinuses normal. Arachnoidal villi moderately developed. Pia mater everywhere transparent and clear except over both hippocampal gyri and the tip of the left temporal lobe. The pia mater is everywhere distended by the swollen brain and contains very little liquid. The cisternae at the base contain considerable slightly turbid liquid. Brain weight 1,165 grams. Convolutions flattened notably on the left side. The cortical surface suggests a moderate pigmentation. The gyri show a normal arrangement and size, in both hemispheres, except for distortion of the left temporal gyri opposite the optic chiasm. This latter process is merely incidental to the distortion of structures consequent upon an aneurysm imbedded in tissue below the left uncus. The aneurysm is globular and measures 1.8 cm. in all directions. The cavity of the dilated vessel is completely filled with

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laminated clot. The wall is of varying thickness, measuring internally, where its surface gives directly upon interpeduncular spaces, from 3 to 4 mm. deep. The remainder of the wall also varies in thickness but rarely exceeds 2 to 3 mm. in depth. The interior of the aneurysm shows chocolate colored clot peripherally with mixed red and gray clot in the middle. The vessel affected is the anterior choroid artery. The proximal portion of the artery shows, in common with the internal carotid and Sylvian arteries in this region, a considerable thickening and dilatation so that the aneurysm appears to be situate upon a kind of yellow stem measuring 1 cm. in diameter. The other arteries at base show well marked focal sclerosis without dilatation, except in the case of the left posterior communicating artery which is dilated to a diameter of 2 mm. and runs in the same axis with the posterior cerebral artery.

The substance of the uncus appears to have been largely destroyed by the aneurysm, but remains of it, together with a portion of the superior temporal gyrus, can be made out in that portion of the brain substance which has been displaced outward and downward by the lesion. The substance of the left putamen, external capsule, claustrum and white matter of the temporal lobe contains numerous dilated perivascular spaces. The lenticular nucleus has been displaced outward and upward but appears to have undergone no diminution in size. The internal capsule has apparently not suffered in width.

The contour of the ventricles appears to be slightly altered by pressure. Frontal section in a plane 1 cm. posterior to the optic chiasm gives the following measurements:

Left lateral ventricle—body
2 cm. laterally x 0.9 cm. from above downwards.
Left lateral ventricle—inferior cornu
2x0.8.
Right lateral ventricle—body
2.3 cm. laterally x 0.5 cm. from above downwards.
Right lateral ventricle—inferior cornu
1.2x3 cm. laterally.

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These measurements appear to show that, whereas both ventricles are moderately dilated, the lateral ventricle on the side of the lesion is somewhat more dilated than its fellow. It is possible that this condition is related with alterations of pressure in the area supplied by the anterior choroid area. Another hypothesis is that the lesion has compressed the foramen of Monro.

The convolutions although flattened are reduced in size and almost of plastic consistence. This character is shared by the cerebellum, pons, bulb and spinal cord. No focal lesions were seen elsewhere about the aneurysm.

The tissues of a case of sixteen years standing cannot afford the striking evidence of acute change which was afforded by Case I. A different sort of evidence is procurable. I have chosen for photographic reproduction the superior temporal gyrus, because that gyrus seems to have suffered most recently from the effects of pressure. (Plate III.)

The photograph shows several points of interest. The normal depth of cortex is well maintained. The innermost layers of cortex are not far removed from normal, exhibiting a few trifling denuded areas. On the other hand, the outer nerve cell layers exhibit a relatively striking irregular denudation of nerve cells. The second cortical layer is notably cut through at one point by a lane virtually bare of the characteristic small nerve cells. And the layer of large external pyramids has preserved but two eyelets of its characteristic cells. Under the stress of a somewhat diffuse pressure, coupled no doubt with arterial changes, the temporal cortex has become somewhat irregularly denuded of a portion of its cells, and these rather in the outer layers. *The larger cells tend to last longest; the smaller cells* (particularly of the second and third cortical layers) *tend to disappear first.*

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It is unnecessary here to linger on this point, which becomes clearer with later cases. I would only insist that not only does inhibition (or hastening action) on the part of these outer layers become more difficult through the absence of whole blocks of cells, but also any impulse once initiated in a process of some intact nerve cell gets a diminishing chance of interruption by countercurrents of any sort. There is a kind of functional vicious circle in tissues subject to these differential lesions. There is a kind of inertia in impulses once set going. Intercurrences are few in this synaptic tissue of lowered quality. The multitude of energy-absorbers which formerly enwrapped the path of a propagating impulse has now vanished. The system is much simplified; and, until such time as the onset of atrophy shall separate the discharging constellations altogether, it would appear that immediate discharges can scarcely be stopped among surviving cells, when they are stimulated.

The present case showed conditions of this sort in all the convolutions abutting upon the aneurysm, and, indeed, slight tendencies in the direction of differential cell atrophy were observable throughout the brain (perhaps the result of chronic internal hydrocephalus.) Such tissues, intermediate between perfect tissues and thoroughly atrophic ones, form the proper soil for keeping up, if not for producing, epileptic discharges.

In Case I we were fortunate enough to light upon an adequate epileptogenic focus in the form of a localized encephalitis in one cornu ammonis. In Case II we again meet with a lesion in one cornu ammonis which may well have formed an epileptogenic focus in the first instance and subsequently formed new ones in the course of its gradual and differential destruction of tissues. In the

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second case the original focus has doubtless wholly vanished with the growth of the aneurysm. All we now find is a medium favorable for unimpeded discharge. The recent active foci may have escaped search; or an epileptic habit may have developed, by virtue of which occasional summations of stimuli from several sources may serve to set off the chain of convulsions.

We now turn to the more debatable ground of those organic cases in which the direct relation of lesions to symptoms is more difficult to prove, largely through the multiplicity of the lesions. (1) By means of Cases III and IV I hope to lend further support to the ideas developed from Case II—namely to set forth more clearly the conception that tissue destruction of moderate degree may frequently leave the discharging apparatus intact, whereas the inhibitory and controlling apparatus is destroyed. We shall find that even coarsely destructive lesions of the motor cortex are prone to this differential effect and that, whether for evolutionary reasons or not, the more delicate portion of the machinery is first lost. (2) Finally in the residue of cases described I hope to develop further the idea of unchecked discharge and a related conception, that of progressive or lateral discharge.

Plates IV and V (from the right precentral and right occipital regions of Case III) show clearly one point: the preservation of large elements and simultaneous disappearance of small elements about small infarcts. Plate IV shows this tissue property most distinctly, because the confusing feature of neuroglia cell overgrowth has passed. The lesion in Plate IV is possibly far older than that shown in Plate V. In both instances, however, there are lanes of nerve cell destruction setting in from the exterior. Along the borders of both lanes the larger elements are

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still in evidence. At the right side of both plates it is evident that the second cortical layer and (to some extent) the third have been destroyed, so that underlying elements are in so far without their influence. At the same time these larger elements (as adjacent sections prepared according to Benda's method show) are now well wrapped with neuroglia (in a quiescent state in the precentral region, in an active proliferating condition in the occipital region.)

Plate IV betrays the stratigraphy of the cortex particularly well with respect to the differential loss of elements of which we are speaking. The outer nerve cell layers tend, so to say, to weather out more readily than do the inner layers when under virtually the same conditions with respect to noxious agent (in this case ischemia.) And, although infarction leaves at times many bizarre figures of differential destruction, yet the configuration of layers seen in Plate IV occurs very frequently in organic cases.

Plate VI (from another case, Case IV) illustrates the same tendency in diffuse atrophy. Again a section is chosen from tissue which is no longer undergoing active neuroglia proliferation, so that the preservation of numerous large elements is manifest, together with a loss of many smaller elements. This appears to be true throughout all the layers; but the outer layers have suffered maximally. Notably in the middle of the layer of moderate sized pyramids, in the midst of a general destruction of all other nervous elements, a single nerve cell of good size remains. The Benda preparation from the adjacent section shows this self-same isolated cell surrounded by the fibrillar neuroglia of the little infarct in which it lies. We cannot doubt both that this cell has lost touch with innumerable streams of impulse from without and that

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also a new surface of separation has been provided for any impulses that may reach the cell.

Under such circumstances not only are the cells divorced from normal controls but they are placed under peculiarly good conditions for direct irritation or for activity engendered by direct pressure.

The details of these two cases follow.

CASE III

G. C., male, born 1840, of average capacity; had a "sunstroke" in the Civil War, and about 1876, while working as customs inspector, had a severe attack of convulsions, which confined him to bed for two or three weeks. There were then no convulsions for several months; but then they recurred and increased in frequency, occurring both at night and during the day, until about 1902. Delirious episodes. Increasing dementia. Unruly. Sent to Soldiers' home, 1903. Incapacitated for work since 1886. Evidence of mental failure since about 1880. Admitted to hospital April 11th. Died June 20th, 1906. Marked insane heredity.

The autopsy (twelve hours after death) showed the following conditions:

General arteriosclerosis (aortic, coronary, splenic, renal, cerebral, iliac, with some gross changes in all large arteries examined.) Calcification in aorta and internal iliac arteries.

Chronic diffuse nephritis (arteriosclerotic.)

Red marrow of femur.

Chronic splenitis.

Loss of cerebral substance in right temporal region, and in lungula, with sclerosis of adjacent white matter and hippocampal gyrus.

General cerebral and cerebellar sclerosis.

Sclerosis of dentate nuclei (maximal) and of olives.

Sclerosis of lumbar cord.

Atrophy of cerebral cortex of frontal poles and of superior vermis of cerebellum.

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Chronic leptomeningitis (vertical, superior cerebellar, and local about loss of substance in right hemisphere.)

Bronchopneumonia with acute fibrinous pleuritis of both lower lobes.

Slight bruises of extremities.

Chronic periappendicitis.

Healed ulcers of stomach.

Chronic gastritis.

Chronic interstitial pancreatitis.

Chronic adhesive pleuritis (right apex.)

Chronic tympanitis.

Following is a description of findings in the head:

Scalp normal; calvarium dense, without diploe. Dura adherent to calvarium over vertex. Dural sinuses normal. Arachnoidal villi moderately thickened. Pia mater of vertex opaque and five or six times as thick as usual, so that the cerebral markings are quite effaced. The pial meshes are distended with clear liquid which stands out in sac-like accumulations over the atrophic frontal poles. The pial thickening is sharply marked off on the flanks of the brain, so that the temporal regions are spared on both sides. The pia mater of the temporal regions and of the base is a trifle more opaque and thicker than normal. The cerebellum and the substantia reticularis alba of Arnold shows the greatest pial thickening found at the base. The pia mater covering porencephalic area in right posterior region is thick and cloudy. Large vessels with primary and secondary branches diffusely yellow and stiff with occasional short stretches of grossly normal tissue. Brain *weight* 1290 *grams*. Substance everywhere firm and elastic. Cerebellum notably firmer than cerebrum. Cortex in superior vermis shows reduction in depth by one-half. Dentate nuclei of cerebellum cut with difficulty and have a leathery feel. Olives perhaps a trifle firmer than usual. Gray matter of frontal poles reduced one-half in depth and a trifle firmer than remainder of cerebrum. Extensive *loss of substance in right hemisphere* under floor of posterior cornua of lateral ventricle. Pia and ependyma here lie in

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apposition over a rounded oblong area 6.5x2 cm. in diameter, extending from the frontal plane posterior to the uncus to a frontal plane 2 cm. anterior to the occipital pole. The loss of substance involves the greater part of the cerebral tissue between the collateral, calcarine, and third temporal sulci. The white matter of the centrum semiovale anterior to this loss of substance is leathery. The hippocampal gyrus on the right is a third narrower and considerably firmer than that on the left. Basal ganglia a trifle firmer than usual. Pons firm, especially near periphery. Cord shows considerable increase of consistence especially of the posterior aspect in the lumbar region.

Middle ears: Free from exudate. Fibrous thickening of drum heads, particularly left.

CASE IV

H. P., female, born 1829, of good heredity, at seventy-two years had a "shock" with cyanosis, convulsions, unconsciousness for some hours, and subsequent speech disturbance and muscular weakness. The convulsions continued at irregular intervals, sometimes several in a day, as a rule several a week, sometimes with remission for weeks. Loss of memory, hallucinations, excitement, supervened at seventy-four. Generalized convulsions at infrequent intervals, especially at night, followed by confusion, paraphasia, complete disorientation, and incapacity to name objects. Death at seventy-six.

The autopsy (three and one-half hours after death) showed the following conditions:

Hemorrhagic infarction of left ventricle of heart.

Aortic, coronary and iliac arteriosclerosis with calcification.

Renal and cerebral arteriosclerosis without calcification, but involving small as well as large branches.

Mitral sclerosis.

Chronic fibrous myocarditis.

Chronic fibrous endocarditis.

Hypertrophy and dilatation of left ventricle of heart.

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Chronic interstitial nephritis.

Bronchopneumonia of hypostatic distribution.

Cholelithiasis.

Congestion of diploe.

General cerebral atrophy (especially frontal and parietal.)

Chronic fibrous leptomeningitis.

Compensatory edema of pia mater.

Small superficial area of yellow softening in left angular gyrus.

Following is a description of findings in the head:

Diploe deep and congested. Pia mater and sinuses not notable. Pia mater over frontal and parietal regions hazy, thickened and distended by considerable fluid. Slight haziness over basal cisternae. Vessels of circle of Willis and primary branches extensively sclerotic. Small cortical branches everywhere prominent. Marked sclerosis of pial vessels overlying small superficial area of yellow softening in region of left angular gyrus. The convolutions of the frontal and parietal regions are narrowed and in places present broad cupping of their surface together with shallow and wide sulci. The brain substance is of normal consistence. The ependyma of the ventricles is smooth.

I present in Plate VII the cell appearances in the orbital region of a confirmed epileptic (Case V.) The reduction in number of elements in the outer layers is striking (comparable to appearances in Plate III and Plate VI.) The atrophy is somewhat general, but, as in numerous instances, the smaller elements suffer first. This section is only an example of what may be found in numerous regions of this case. Palpable sclerosis was confined to a part of the right central and frontal regions.

The details follow.

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CASE V*

A. H., negress, born 1860. Convulsions appeared in the patient's early thirties and were frequent for a month. They returned again at thirty-nine years but occurred at rather infrequent intervals. Remarkable opthalmoptosis, giving rise to the suspicion of brain tumor. Vision fairly well preserved. Opthalmoscopic examination showed only swollen veins. Headache. Increasing weakness. Slight external strabismus (left). Left knee-jerk slightly brisker than right. Impairment of memory. Emaciation. Conjunctivitis. Hypopyon. Death in coma at the age of forty-six. No convulsions were noted during the last two months. Irregular course movements of arms.

The autopsy (eleven hours after death) showed the following conditions:

Encephalomalacia and myelomalacia.

Early bronchopneumonia left lower lobe.

Sclerosis of middle two-thirds of right central convolutions and posterior part of right middle frontal convolution.

Atrophy of left optic nerve.

Conjunctivitis, keratitis and hypopyon of left eye.

Left opthalmoptosis.

General arteriosclerosis (aortic, coronary.)

Slight mitral sclerosis.

Old infarct of kidney.

Chronic external adhesive pachymeningitis.

Chronic adhesive pleuritis.

Chronic focal adhesive pericarditis.

Mural and subperitoneal fibromyomata of uterus.

Following is a description of findings in the head:

Scalp edematous anteriorly. Calvarium thick, with little diploe. Dura adherent to calvarium in bregmatic region. Sinuses normal. Pia mater edematous, without notable thickenings. Brain weight, 1,055 grams. Substance remarkably and evenly reduced in consistence in

* Published more fully in a "Note on Cell-Findings in Soft Brains," by E. E. Southard and M. B. Hodskins, American Journal Insanity.

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both white and gray matter. Olives and dentate nuclei as soft as normal cortex cerebri. The appearances resemble those of many days post mortem. The central convolutions of the right hemisphere are much firmer than the surrounding convolutions. The right ascending frontal convolution is firm throughout the arm area and for a portion of the face and leg areas. The right ascending parietal convolution is equally firm for a like distance. The sclerosis is sharply marked off by sulci, by the post-central sulci behind (so as to exclude the superior parietal and supra-marginal regions) and by the anterior ramus of the inferior precentral sulcus and the superior frontal sulcus in front and above (so as to exclude the greater part of the middle frontal and the superior frontal gyri). There is no demonstrable atrophy or chronic pial reaction in relation with the sclerosis. The white matter beneath presents no change except the universal reduction in consistence.

Cord: Numerous calcified plaques in posterior lumbar pia.

Middle Ears: Left middle ear contains semi-liquid pus.

Retinae removed from behind show left nerve-head reduced about one-third in surface area. Vessels injected.

Plate VIII exhibits the right precentral (leg) area in an epileptic of long standing (Case VI). *There is marked atrophy of the smaller elements with maintenance of the great motor cells intact.* As the autopsy report shows, there was extensive sclerosis in many parts of the nervous system. Yet the motor cells remain, forming parts of an extremely simplified series of arcs.

CASE VI

W. R., male, born in 1854, began to have convulsions at seven years of age after whooping cough. The convulsions were severe, averaged one a month, and ceased to occur about 1884. About 1899 the convulsions reappeared

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and dementia became pronounced. The patient was admitted to hospital January 15, 1906. The left leg was found somewhat smaller than the right, whereas the arms were equal. The toes of the left foot were extended at the first joint and flexed at terminal joint. Instability in Romberg position. Left leg weaker than right. Vision of right eye diminished. Slight internal strabismus. Pupil reactions normal. Knee-jerks lively. Left knee-jerk and Achilles jerk more lively than right. Babinsky of left side. Stupid, untidy, grimacing, snarling and irritable. In February his mood changed to a laughing childish dementia. Convulsions appeared in April. Gradual failure. Death December 28, 1906.*

The autopsy (twenty-three hours after death) showed the following conditions:

Occlusion of larynx.

Congestion and edema of lungs.

Chronic diffuse nephritis.

Congestion of kidneys.

Congestion of mesenteric veins.

Calvarium dense.

Chronic external adhesive pachymeningitis.

Chronic fibrous leptomeningitis over left superior parietal and left central gyri, hippocampal gyri, and superior vermis of cerebellum.

Sclerosis of dentate nuclei.

Sclerosis of pons.

Sclerosis of hippocampal gyri, of orbital regions, of precentral gyri, of post-central gyri, of posterior part of superior parietal lobules, of fusiform lobules (slight.)

Sclerosis about posterior horns of lateral ventricles.

Anomalous fissuration of superior vermis of cerebellum.

Following is a description of findings in the head:

Hair short, streaked with gray, thin on top. Scalp normal. Calvarium shows no diploe. Dura adherent over occipital and superior parietal regions where, notably

*I am obliged to Dr. H. W. Mitchell for these notes drawn from the records of his Danvers service.

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on the left side, there is a cluster of villi surrounded by moderately thickened pia mater. Pia mater thin and transparent everywhere except along lip of longitudinal fissure on left side in region of arachnoidal villi and over left central convolutions, along inner borders of both hippocampal gyri and over superior vermis of cerebellum, especially in the region of the clivus monticuli. The posterior wall of the cerebellopontine cisternae and the pia mater about the surrounding cisternae in the region of the optic chiasm are moderately dense and suggest no notable degree of fibrosis. Vessels show nowhere any trace of sclerosis or lesion. The veins of the vertex are moderately injected but the small vessels over the surfaces of the gyri do not stand out prominently. Brain weight, 1,290 grams. The brain substance shows striking varieties in consistence. The laminae of the superior half of the cerebellum, including the vermis, show the best marked loss of consistence anywhere shown; and this loss of consistence appears to be sharply limited posteriorly by the horizontal fissure. On section this loss of consistence proves to be superficial and the white matter of the arbor vitae is as firm as elsewhere. The dentate nuclei show an increase of consistence in both hemispheres. The posterior third of the pons is softer than the anterior two-thirds; the difference is probably due to gliosis of the anterior two-thirds. On section the pons appears to show some graying out in the central regions superior to the pyramidal tracts. The central gray matter is far softer than the rest of the pons. The cerebral hemispheres show palpable in durations, (1) of both hippocampal gyri, (2) of both orbital regions for a space 1 cm. in diameter surrounding the olfactory bulbs, (3) the upper two-thirds of both precentral gyri, best marked on the left side where the induration extends a trifle posteriorly to include the post-central convolution, (4) both post-central gyri in less degree than in precentral, (5) both superior parietal gyri in their posterior halves, (6) in slight degree both fusiform lobules. The maximal palpable induration is found

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in the superior part of the left precentral gyrus and in the two cornua ammonis where they project into the ventricles. The dentate nuclei of the cerebellum are almost as firm as the regions just noted. The lateral ventricles contain little fluid and show no ependymitis. There is slight induration of white matter surrounding the tips of both cornuae. The basal ganglia show no evidence of lesion. The two optic thalami appear of nearly equal size; the left globus pallidus is slightly yellower than right. The cerebellum measures 10.8 cm. in greatest lateral diameter, the superior vermis measures 5 cm. to the cerebellar notch. The cerebellar notch appears a little deeper than usual on account of encroachment of fossa which hollows out the posterior half of the superior vermis, halving especially the clivus monticuli. This fossa is smooth-walled and overlaid by a moderately thick layer of fibrous tissue. The fissural markings of the left lobus clivi do not run concentrically about into their fellows of the right lobus clivi. The left lobus clivi is divided into three sets of folia separated by deep fissures. The superior set of folia clivi dips sharply upward in the hollow of the anomalous fossa of the clivus monticuli so as to become continuous with the middle set of folia in the right lobus clivi. The appearance is thus presented of sharp termination of the superior set of folia in the right lobus clivi interiorly. Sagittal sections show corresponding disturbance of the fissuration. A section slightly to the left of the median line shows that the folia usually exhibited in the clivus monticuli are replaced in a small area not over 1 cm. in diameter by the flat non-foliated tissue. Adjacent sections rapidly regain the normal fissure appearance. This lesion may be interpreted as a loss of substance of long standing. The overlying fibrosis may perhaps be interpreted as a reaction to the original injury. Spinal cord: fairly firm. Cervical region softer than usual. Lumbar portion firmer than usual. Middle ears: normal. Pituitary body congested.

Plate IX is also derived from Case VI, but may be con-

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sidered together with Plate X from Case III as showing similar phenomena. Attention is here directed to remarkable gaps of an atrophic character in the closely set cells of the stratum granulosum of the cornu ammonis. The lesion in Case VI was nowhere found related with vessels. The focus shown in Plate X (Case III), like foci in other sections from the case, seems related to the presence of a vessel. These atrophic foci are, as adjacent sections stained by the Benda method demonstrate, attended by considerable gliosis.

Such changes are by no means confined to the cornu ammonis in cases of epilepsy, but in the close search of his cases made by a worker in epilepsy such changes are not rarely found. The archipallium is thus the scene of certain lesions of the receptive apparatus whose meaning we cannot yet make out. In the archipallium, although we have to do with similar (in the sense of less coordinated, less inhibitory) mechanism, we also have to do with more compact structures in which small lesions may bring about relatively more irritation than lesions of the same volume set anywhere in the higher apparatus. This may serve as a suggestion why cornu ammonis lesions are relatively so apt to the production of powerful irritations. In Case I we dealt with an effective epileptogenic focus of small diameter; but it is possible that other cases might yield still smaller foci, were methods of examination fine enough.

In the above selection of cases I have brought out certain points with undue clearness, with the object of supporting certain contentions of a general character. Whether or not the detail of these inductions is in all ways accurate, I hope that it is plain that the classical fiber-tract studies will not solve the problem. I have, however,

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steered clear of making any inductions from neurofibril preparations because, as is well known, positive pictures of neurofibrils have far more convincing power than negative pictures. The problem, I believe, rather resembles that of multiple disseminated sclerosis than any other with which I am acquainted. The natural history of gliosis, were it completed, would go far to settle many problems, but particularly these.

I have therefore employed for the nerve cells the best general method for their demonstration that I know—the original method of Nissl (with very few and quite inessential modifications)—and have obtained an adequate picture of the neuroglia *in adjacent sections* by the Benda method for neuroglia (after alcohol fixation). In this way it is possible to obtain a better conception of mechanical conditions surrounding cell-groups than by any other methods known to me. Moreover, these methods are quite simple and repeatable. The Nissl method is not precarious or capricious. The Benda method, though perhaps inferior to the Weigert and the Mallory methods under the best conditions, is wholly adequate for pathological neuroglia in most cases, and has the tremendous advantage of being *applicable to sections adjacent to the Nissl sections.* Nevertheless, I believe the important points of the present paper could probably all be made with far simpler methods, with any method which stains cells well and preserves the stratigraphy of the cerebral cortex.

SUMMARY

The theory of epilepsy expounded in the present paper is founded mainly upon structural considerations. The histological data have been interpreted largely from a functional point of view. The theory lays claim to some originality in two directions, in setting forth, namely: the

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properties of a typical epileptogenic focus in the cerebral cortex and the nature of that change in cortical tissue which favors epileptic discharges. The characteristic feature of a typical prime focus is described as the separation of a normal cell-group from its normal control by other cell-groups and the impact upon the receptive surfaces of these normal cells of a steady, intimate, abnormal pressure—both segregation and compression effected by neuroglia overgrowth. That feature of cortical tissue which favors the spread of epileptic discharges is described as due to a simplification of cell arrangements, arising in the destruction of controlling elements with maintenance of motor elements. In the production of both prime focus and the abnormal tissue which permits uncontrolled discharge, the neuroglia tissue plays a characteristic part—exerting an active continued pressure in the first instance, and readily permitting lateral discharges and the activation of great groups of motor cells in the second instance. In the former case we see a fresh example of the irritative property of heightened tension—only here exhibited quite in miniature. In the latter instance we are dealing with conditions of still greater theoretical interest, approximating, though with diverse outcome, the loss of insulation seen in foci of disseminated sclerosis. The findings suggest the widely different effects upon nervous tissues of active and of quiescent gliosis.

From a review of pertinent literature, it appears that physiological interest is converging upon the field here considered. Fiber-tract studies have failed to cope with other problems than those of linear transmission along well insulated paths. Only in the case of multiple disseminated sclerosis and certain studies in interstitial neuritis, have the occurrence and nature of lateral dis-

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charge from fiber to fiber and the effects of intimate fiber pressure been considered. And in these instances it may well be proposed that a fresh abnormal type of synaptic tissue has been provided. Physiological interest is now levelled upon the synaptic tissues in general. And, if a synapse is a physical surface of separation between neurones, it is serviceable to inquire what are the conditions which can readily modify the synapse. The neuroglia tissue, formerly regarded as purely supportive in function, here rises to a high scale of importance. The present essay points out two effects of gliosis upon synaptic tissues—the one an active irritative one, the other a passive effect. A review of the fundamental views of Hughlings Jackson serves to demonstrate the perfect generality of epileptic phenomena at all levels, and makes clear why the writer sought knowledge about epilepsy in organic cases. If the writer advances a case in which the prime epileptogenic focus consists in an active gliosis within a space of one cubic centimeter in the cornu ammonis, he cannot be charged with holding that all cases of epilepsy are so brought about. He describes what he regards as a typical prime focus. He conceives fundamentally that similarly forcible and lasting stimulation of a receptive surface, standing in important relations to the motor system, might produce epileptic convulsions just as effectively as the gliosis he describes. In this sense, complex emotions or intestinal worms might conceivably stand in as effective a relation to the nervous system as the intimate pressure of early gliosis upon the expansions of elements whose currents eventually play upon the muscular system.

Wholly distinct from these considerations about epileptogenic foci are those points which are developed concerning tissues facilitating discharge. A review of various

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authors discovered much difference of opinion and considerable interpretation of phenomena as secondary. The phenomenon of gliosis has not escaped numerous observers, among them the very observers that have emphasized the alterations of the second cortical layer as important in epilepsy. But this gliosis has been regarded as secondary, and our attention has been diverted rather to certain cell and nuclear characters which are looked upon as specific. The writer has been tempted to regard these nerve-cell changes as vegetative and at any rate as not further analyzable, but to accept them as examples of a lesion which will interfere with normal control of muscular elements. The cases presented here go far to prove that the nerve cells of the outer layers are the first to disappear in cases of atrophy and even along the edges of ischemic areas. The tendency to the formation of tissue favorable to epileptic discharge is, according to this view, a somewhat general tendency in cerebral tissue, so long as the destroying forces stop short of the motor elements and permit any communication, however slight, between the motor elements and the receptive side of the body. A reduction or simplification of the system through destruction of the smaller elements of the cerebral cortex procures new reflex arcs with fresh surfaces of separation which are perhaps even simpler and more automatic than the spinal arcs and synapses. The peculiar features of the epileptic discharge depend upon the inertia of currents traveling in simplified arcs, and upon the lack of energy-absorbents en route. The cerebral arcs normally escape automatism through a multitude of synaptic connections; under epileptic conditions the cerebral mechanism approaches in fatality the spinal mechanism.

Under this conception epilepsy and phenomena like clonus

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are readily perceived to belong to a single logical group. The phenomenon of epilepsy, in short, requires the intactness and even the normality of some well-defined route from stimulus to muscles. If we conceive the stadia of this route set end to end with the cerebral synaptic tissue in the middle, we perceive that towards the two ends of the linear series it becomes increasingly difficult to provide conditions which will produce generalized and spreading convulsions. Destruction of elements at any point in the route should at first sight exclude the production of epilepsy. And so, in most cases, the destruction of the efferent paths will exclude epilepsy. In the afferent paths, however, the very process of destruction often constructs new and potent surfaces of stimulation which act as epileptogenic foci; and, in the cerebral synaptic tissue, the strata are so constructed that the loss of smaller, central, modifying, and inhibitory elements is effected prior to the loss of the major elements which are essential to the intactness of the great route. And these major efferent elements can themselves be subject from time to time to stimulation afforded by the contractile energies of growing neuroglia. Epileptogenic stimuli are applied in all cases to those elements having a forward direction, so that the reaction is in most cases, if not necessarily, a sensorimotor reaction in Hughlings Jackson's sense.

What are the applications of this theory to the phenomena of idiopathic epilepsy? In certain cases of idiopathic epilepsy there seems to be grave doubt whether any adequate epileptogenic foci can be discovered. There is more hope that *tissues favorable to epileptic discharge* shall be discovered, if the proper methods are employed.

We can see some reason for the absence of effective foci in hereditary cases, particularly if we bear in mind the

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epileptic offspring of Brown-Sequard's guinea pigs. With the onset of topographic and stratigraphic knowledge of the cerebral cortex, we shall approach more nearly to a definition of tissues suitable for the propagation of epileptic discharges. So far it seems that such synaptic tissues are characterized by abnormally simplified arcs whose impulses are the more automatic through the lack of countercurrents from surrounding cells. Whether we are to look in inherited serum properties for the production of such conditions, the future will decide. Destroying agents of moderate power tend to alter tissues in this direction.

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PLATES

All the photographs, except Plate I, have a magnification 1:50; Plate I, 1:40. All the sections, except those photographed for Plate I, were prepared and stained according to the method of Nissl (without essential modification). The sections photographed for Plate I were prepared from blocks fixed in Zencker's fluid and were stained according to Mallory's phosphotungstic acid hematein method for neuroglia.

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PLATE I.

FOCAL SCLEROSIS IN (A) PLEXIFORM LAYER; (B) PERIVASCULAR REGION FROM PRECENTRAL CORTEX IN A CASE OF ORGANIC EPILEPSY ARISING IN OLD AGE. Such scleroses, though frequent in certain cases of epilepsy, are not regarded as bearing in any important way upon the problem of epileptic discharges.

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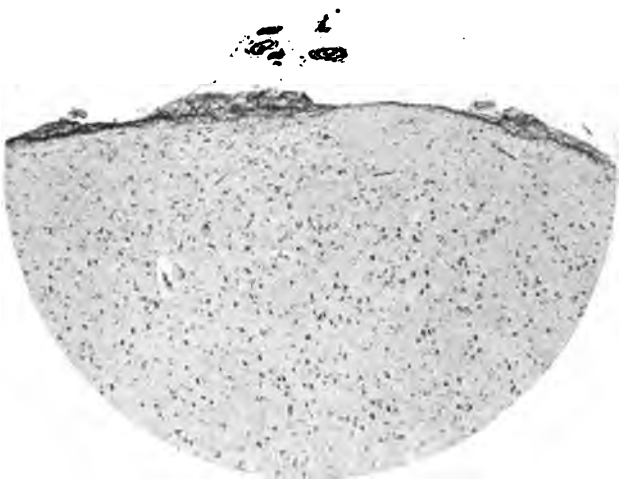

FIG. 1

FIG. 2

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PLATE II.

ACUTE ENCEPHALITIS FOCAL IN ONE CORNU AMMONIS IN A CASE OF EPILEPSY LASTING NINE WEEKS AND TERMINATING IN STATUS. The great nerve cells are still intact, but between the nerve cells are great numbers of proliferating neuroglia cells, amounting externally to a dense black zone. As a bit of intact nervous apparatus under abnormal pressure conditions, the cells of this area—nowhere greater than 1 cm. in diameter—are capable of acting as an EPILEPTOGENIC FOCUS. A severer lesion might destroy the focus either directly or by cutting off the transmission of impulses toward the motor apparatus.

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PLATE III.

DIFFERENTIAL ATROPHIC PROCESS INVOLVING SUPERIOR TEMPORAL GYRUS IN A CASE OF ANEURYSM OF ANTERIOR CHOROID ARTERY, PROBABLY OF SIXTEEN YEARS' DURATION. Other gyri in both hemispheres showed evidence of similar differential atrophy. The SECOND CORTICAL LAYER is cut by a lane of sclerosis at the left hand. But two appreciable islets of large cells remain in the LAYER OF LARGE EXTERNAL PYRAMIDS. *The inner layers are fairly well preserved.* Compare particularly Plates VII and VIII for similar reductions of a differential character in other gyri and other cases. Compare Plates IV and V for theoretically similar reductions in cases of multiple miliary infarctions of the cortex.

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PLATE IV.

SMALL OLD INFARCT OF PRECENTRAL CORTEX (trunk area) IN CASE OF ORGANIC EPILEPSY. Characteristic differential destruction of layers in which the SECOND AND THIRD CORTICAL LAYERS SUFFER MAXIMALLY. The larger cells appear more resistent, (compare particularly Plate VIII for a theoretically analogous loss of smaller elements with maintenance of great motor cells of the leg area in a case of generalized atrophy.) The denuded area is filled with neuroglia tissue, containing relatively few nuclei and no longer exerting active pressure upon nervous elements. Synaptic tissue such as here shown possesses fewer modifying or inhibitory small elements than normal and is a tissue appropriate for epileptic discharges just in so far as the preserved elements have a "forward direction" of impulse (musclewards).

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PLATE V.

(Same case as shown in Plate IV). EARLY INFARCT of occipital cortex with denuded area filled with neuroglia cell nuclei. The preservation of many large cells at the borders of the infarct is noteworthy (compare Plate IV). The infarct is still capable of exerting active pressure upon the neighboring nerve cells, and this pressure is theoretically capable of serving as epileptogenic agent after the manner of the lesion shown in Plate I. The neuronic links having "forward direction" toward the motor region must, however, remain intact. The combination of such active (pressure-exerting) foci of sclerosis with older foci (like that in Plate IV) which have destroyed much of the inhibitory apparatus, must favor epilepsy.

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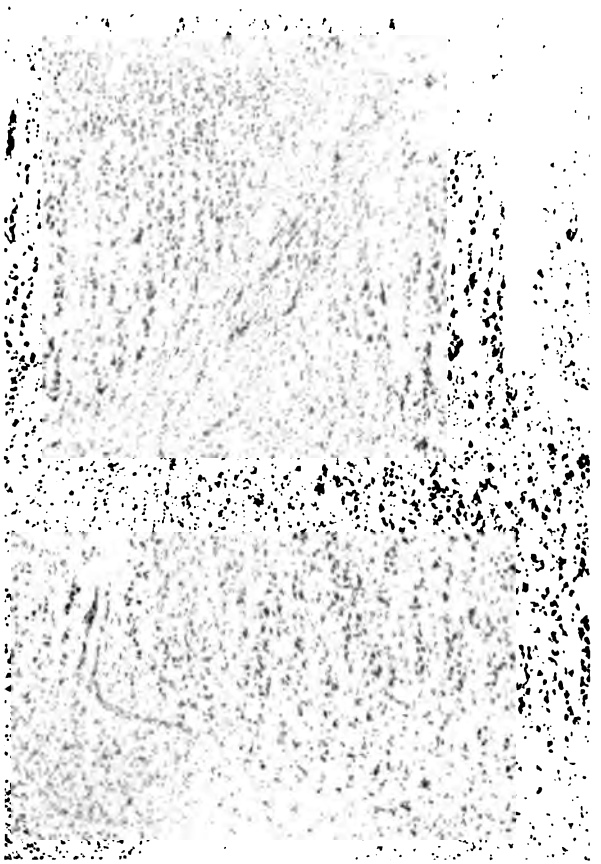

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PLATE VI.

A Second Case of Epilepsy Showing Multiple Small Infarctions, illustrating similar considerations to those of Plates III and IV.

1. Atrophy more marked in outer layers.
2. Maintenance of layer cell within borders of a small oval infarct.

(The Benda preparation of an adjacent section showed this *normal cell imbedded* in *fibullar neuroglia*. Here is offered the theoretical possibility of impulses of an epileptogenic character set up directly in efferent elements. The more frequent finding of epileptogenic foci in "sensory" or receptive parts of the central mechanism is perhaps due to the greater extent of these mechanisms.

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PLATE VII.

ORBITAL CORTEX IN A CONFIRMED EPILEPTIC WITH MENTAL SYMPTOMS (epilepsy over ten years in duration). This area, in common with numerous other cortical types, showed a diffuse atrophy affecting most markedly the outer cell layers. Compare Plates III, VI, VIII.

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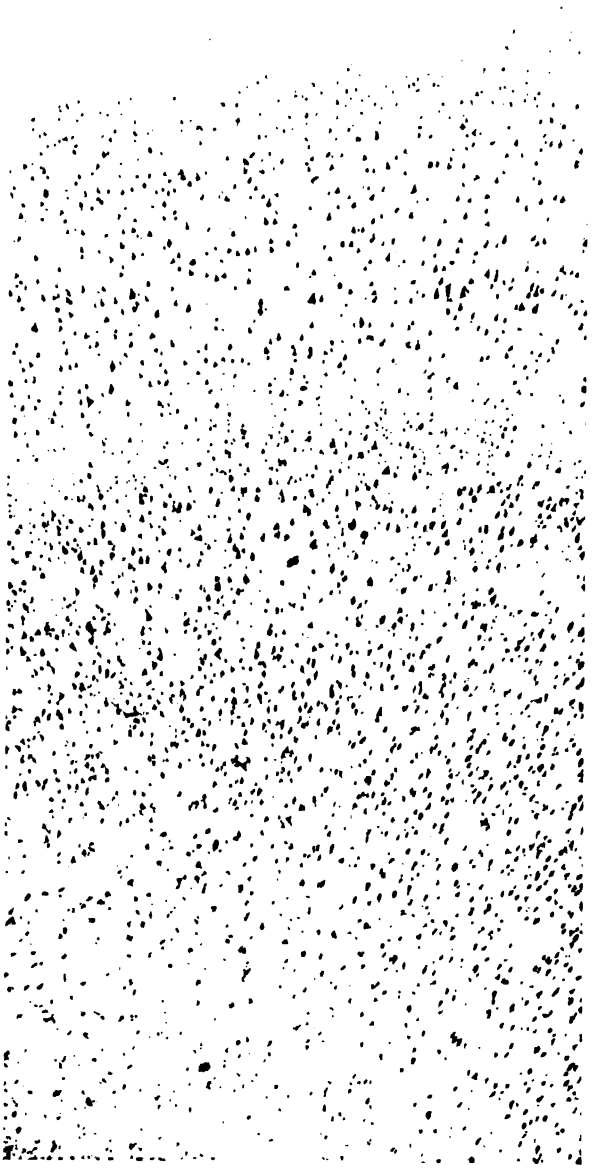

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PLATE VIII.

DIFFUSE ATROPHY OF PRECENTRAL CORTEX. NOTABLE PRESERVATION OF LARGER ELEMENTS. SIMPLIFIED OR REDUCED SYNAPTIC TISSUE SUITABLE FOR AUTOMATIC DISCHARGES IF STIMULI ARE PROVIDED.

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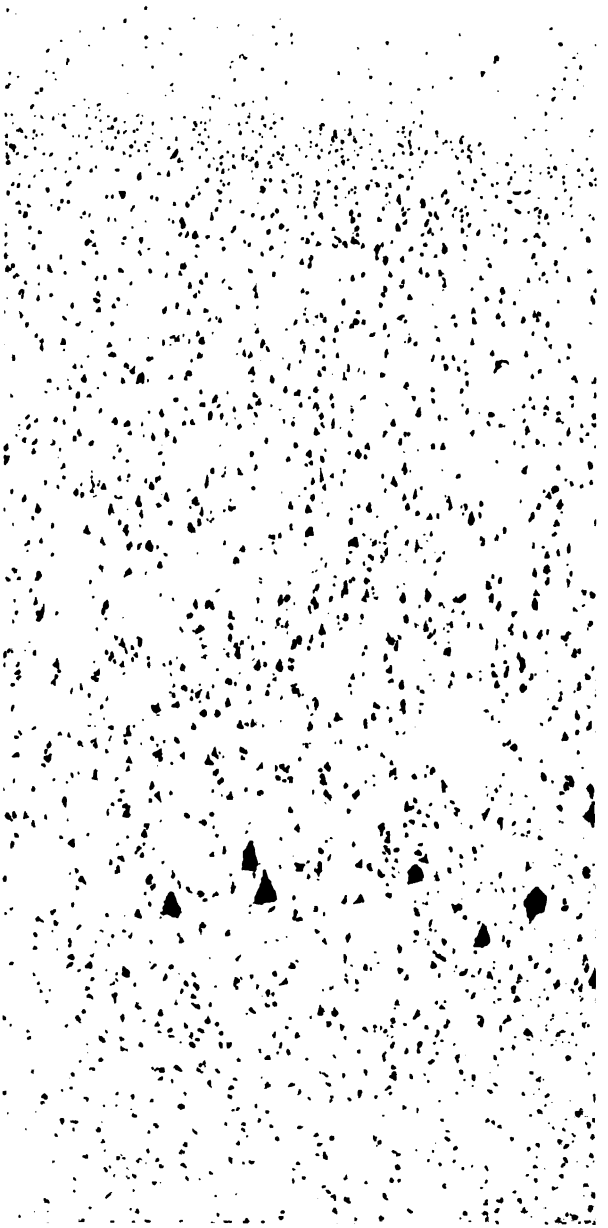

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PLATES IX AND X.

CORNU AMMONIS IN TWO CASES OF EPILEPSY. MINOR ATROPHIC LESIONS INTERRUPTING GRANULAR STRATA. In Plate X one of the gaps is probably related to a vessel about which the benda preparation shows an inactive gliosis. Such lesions frequently occur in epilepsy, but are sometimes found in non-epileptic cases. Early in their formation these lesions may possibly serve as epileptogenic foci, but this can not be proved. The same conditions which produce them are found elsewhere in the cerebral cortex, producing the milder differential lesions which render the tissue suitable for epileptic discharges. Plate IX comes from the same case as Plate VIII; Plate X from the same case as Plates IV and V.

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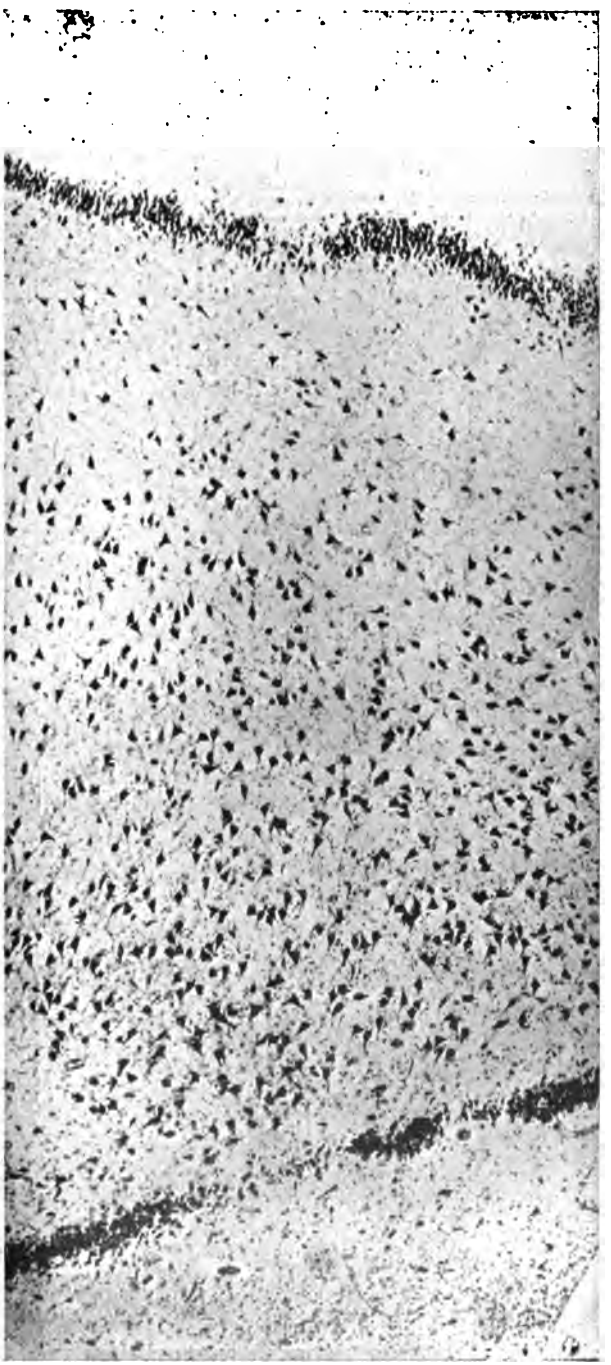

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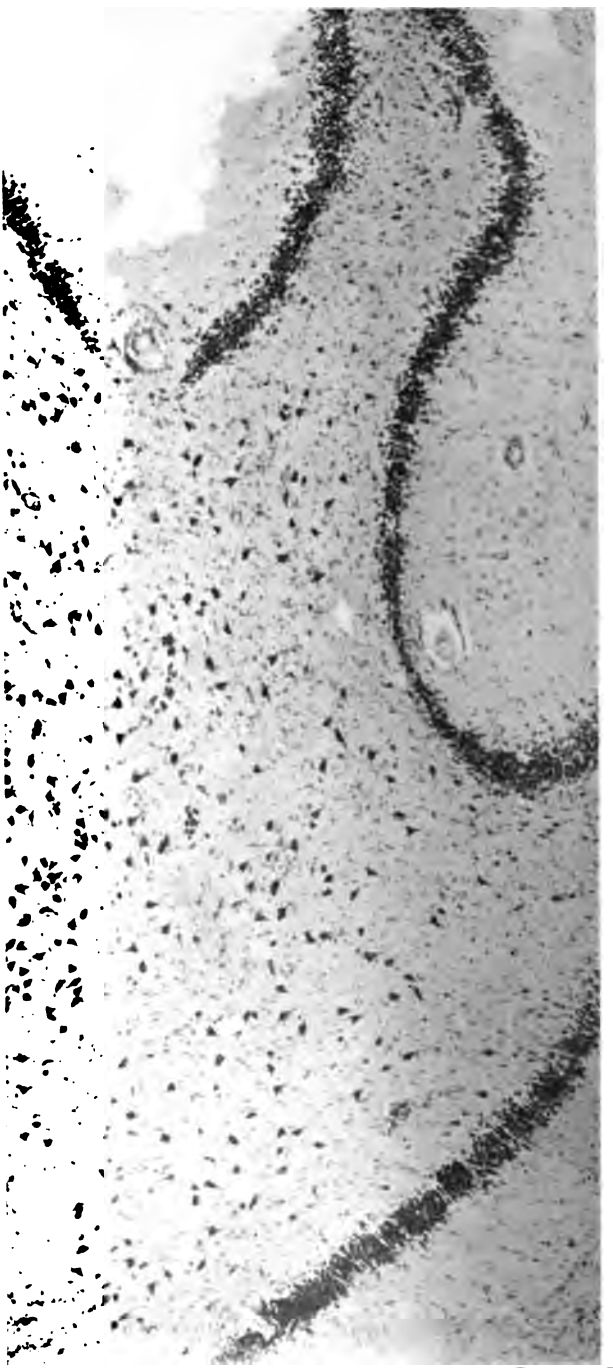
PLATE X



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PART II

REPORT ON THE PUBLIC CARE OF EPILEPTICS
FROM STATES, FOREIGN COUNTRIES AND MUNICIPALITIES

NOTE: Much of this data was read at the Richmond, 1907 Meeting of the Association.

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REPORT ON THE PUBLIC CARE OF EPILEPTICS FROM STATES, FOREIGN COUNTRIES AND MUNICIPALITIES

REPORT FROM AUSTRALIA

By Alice Henry

The conditions of life in America and in Australia are in many respects so similar and the problems to be attacked in these two new countries so often present themselves under similar guise, that it is earnestly to be hoped that in the future there may be closer relations between workers for improvement, both on the official and non-official basis. Progress on various lines is notoriously uneven and where the elder sister among the British speaking offshoots has forged ahead, the other has sometimes lagged behind, as on this epileptic question; whereas in the case of neglected children and in the even larger sphere of maintaining fair conditions of remuneration and conditions for the great bulk of her people, Australia and New Zealand are leading the world today.

It is with deep gratitude that I acknowledge on behalf of my people the aid we have obtained from our fellow workers in England and in America in the task of collecting information on the Colony treatment of epilepsy.

In Melbourne, Dr. J. Y. Fishbourne and Mr. Thomas Harlin were among the earliest to insist upon the colony care for epileptics and upon the responsibility laid upon

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the community of providing such care. It was long, however, before the public could be induced to see the matter in this light. The Austin Hospital for Chronic Diseases, of which Mr. Harlin was Treasurer, made the first tentative public appeal seven years ago, in the form of a suggested annex to the institution. This came to nothing and the matter rested for some time. Meanwhile I had the honor of reading a paper upon "Farm Colonies for Epileptics" at the Hobart Conference of the Australasian Association for the Advancement of Science. In October 1903, the National Council of Women, headed by their President, Lady Sydenham Clarke, took the matter up, urged on by Dr. E. Mary Page Stone and Miss Catherine Helen Spence of South Australia, that veteran worker whose eighty years seem to have no effect on her other than a maturing of her judgment. Her "State Children of Australia" just published is likely to be a handbook for many years to come.

The result of these efforts was the formation of a committee including Dr. Fishbourne, Dr. Springthorpe, Dr. Stawell, Dr. E. M. P. Stone, Mr. Prout Webb, of the Lunacy Department, Captain W. Davis, of the Neglected Children's Department, Mr. R. W. L. Ievers, Dr. Douglas Shields, Lady Clarke, Lady Madden (wife of the Chief Justice) Mrs. Joseph Saddler, Miss M. G. Cuthbertson, assistant inspectors of factories, Mrs. Evelyn Gough and the writer.

March of this year saw the Talbot Epileptic Colony opened, the first institution of the kind in the Southern Hemisphere. It takes its name from Lady Talbot, wife of Sir Reginald Talbot, who in the meantime had succeeded Sir George Sydenham Clarke as State Governor. She has all along taken the keenest interest in the movement. It

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is in a retired and beautiful spot at Clayton, on the rolling, heathy downs that overlook the bay. The grounds, 165 acres in extent, were the gift of Mr. James Mason. The administration block occupies the center and the attached cottages are for men and women respectively. Accommodation is provided for seventy patients. Thirty-five thousand dollars has been spent on the building and furnishing. The money was collected by public subscription, supplemented by a generous gift from the government, and another from the proprietors of the "Argus."

The managers anticipate being able to carry on general mixed farming successfully, especially as the climate is so mild that out-of-door work can be carried on all the year round.

There are many administrative problems which will have to be solved with reference to local conditions on the spot. The question of the admission of patients will be one of the most difficult as it is one of the most pressing. The more conservative on the committee urge moving slowly and cautiously, since it is those least suitable for colony life whose friends always clamor for permission to place their disabled relatives in such a home. The institution has another function to fulfill beyond *sheltering* its quota of patients. It is the pioneer and will probably serve as the prototype of similar colonies as they are established elsewhere in the Commonwealth. It is showing the direction to which State effort will have sooner or later to take.

But with much of our practical work in administration yet to learn, we are deeply thankful to have arrived at the point of having a Colony and I ask your congratulations for my countrymen upon the success of their efforts.

71 Park Avenue, Chicago, Ill.

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A MOTION

After the reading of Miss Henry's communication, it was moved and carried that the congratulations of the National Association upon the successful outcome of their efforts, be tendered to the workers in Australia.

SWITZERLAND

The Swiss Hospital for Epileptics is located at Zurich, Switzerland, and was founded August 31, 1886. It has a population of 201 persons, 119 men and 82 women. The estimated epileptic population of the country is 10,000,—about three per cent of the entire population. It is expected that the Colony will in time accommodate 350 patients. At present a building is being prepared to hold ten men patients of the higher grade, and there are plans for an isolation building for infectious diseases. All persons above seven years of age are admitted, except those who are too insane for an open institution. The Colony is supported out of the allowances of the institution, by farming and gardening, by interest on the capital and by voluntary gifts. The per capita cost for the last fiscal year (yearly) was f. 855. There is an endowment fund of f. 500,449.95. The chief industries are farming, gardening, tailoring, sawing and splitting of firewood, shoe-making, and sorting of silk, the value of these industries for the last fiscal year being f. 26,057.80. Schools are maintained, fifty-one patients attending. There is one physician on the staff besides the Medical Superintendent.

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A laboratory is maintained. The minimum and maximum wages paid men attendants are f. 400—f. 10,000 (probably including physicians;) women f. 350—f. 600. It is stated that about eight per cent to ten per cent of epileptics can be cured.

KANSAS

The Kansas State Hospital for Epileptics is located at Parsons, Kansas, and was founded in 1903. It is laid out somewhat on the village plan, and has a population of 380. The estimated epileptic population of the State is 3,200. It is expected that the institution will grow to have a population of 800 or 1,000 patients. At present a deep water well is being sunk, and a $15,000 water plant will be installed as soon as the well is completed. Contracts will soon be let for administration building and assembly room to the extent of $70,000. Both sane and insane patients are admitted; in fact, any citizen of the State who is epileptic, except low grade imbeciles, idiots and very young children. The Colony is supported by the State, the per capita cost for the last fiscal year being $180.57 (yearly.) Patients are committed by Probate Court and on voluntary commitment. The chief industries are farming and gardening and their estimated value for the last fiscal year was $5,084.92. Schools are maintained, about forty patients attending. Besides the Medical Superintendent, there are two physicians on the staff. There is a general clinical laboratory maintained. The

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minimum and maximum wages paid men are $23-$35; women $20-$30. In regard to the curability of epilepsy, it is stated "some epileptics are curable; if sent to institutions in early stages, ten per cent to fifteen per cent; if kept at home until chronic (as is the rule at present) not more than three per cent."

PROVINCE OF ONTARIO

The Oxford Hospital for Epileptics, located at Woodstock, Ontario, was founded in April, 1906. The present capacity is eight, and two cottages containing room to accommodate one hundred are approaching completion. It is expected that the institution will in time accommodate eight hundred patients, there being eight to ten hundred epileptics in the Province. All sane epileptics above six years of age, for whom there is reasonable hope, are received as patients. The institution is supported by the Government, and it is stated that: "All patients are supposed to pay $3.00 per week." Commitment is by physician's certificate. The chief industries are farming and gardening, and during the last fiscal year were valued at $3,000. Epilepsy is regarded as curable in possibly five per cent of the cases. The medical work is in charge of the Medical Superintendent and one physician. Male attendants receive $22 to $30 per month, and women from $15 to $18.

J. J. WILLIAMS,
Medical Superintendent.

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DR. J. J. WILLIAMS OF ONTARIO:

We experience one difficulty that is unfortunate. After our patients begin to show improvement, their friends or relatives interfere and wish to take them from the Colony. We wish the Legislature to pass a law forbidding parents or laymen from removing patients from the institution without our consent. It is for us who are trained in the work to judge of the best interests of the patient, and not the layman.

We hope for good results at our Colony, and shall spare no effort to obtain them.

CONNECTICUT

The situation in Connecticut regarding the establishment of a colony for epileptics remains unchanged in so far as any material assistance from the State, as represented in our recent legislative body, is concerned.

The committee began its work in good season, and for the purpose of securing as much information as possible, visited the colonies in New York, Massachusetts and New Jersey, where the practical workings of the institutions were observed in detail. In addition to being courteously received, we were placed in possession of valuable material which served admirably in promoting the interests of our epileptic class, when we were confronted with the inquisitorial bias so common to legislative committees.

Dr. Everett Flood, of the Massachusetts colony, was present at the first hearing of our bill, and rendered

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notable service in setting forth, in a clear and concise manner, the necessity for separate institutions for epileptics, from social as well as economic points of view.

In addition, our committee had secured other eminent medical as well as legal talent, and made a strong and convincing presentation of our claims for the passage of the bill. We were encouraged when the committee before which our bill was first heard made a special visit to Dr. Flood's institution, and returned enthusiastic over the colony system, and with the expressed assurance of its co-operation when the time should arrive when we would be called before the committee on appropriations. It was before this latter committee that we met our Waterloo. Very short notice was given of the hearing, and this item alone made it impossible to secure valuable assistance promised in time for the date announced by the committee. Notwithstanding many drawbacks, a goodly array of speakers was present, and unanswerable arguments in favor of an institution were offered, but to little purpose. All that was accomplished was the assignment of the bill to the next General Assembly which holds its session in 1909.

We deeply regret the outcome of our efforts in behalf of the epileptic, but will continue the work already begun with renewed energy, and hope for more favorable results when the time arrives for us to appear before those who have the power to recommend or reject our petition for promoting the interests of this class of cases by establishing a suitable institution for their intelligent care and oversight.

September 1, 1907. DR. E. A. DOWN.

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PENNSYLVANIA

The Eastern Pennsylvania State Institution for Feeble-Minded and Epileptic at Spring City, Pa., was founded May 15, 1903, and is not yet opened to receive patients. Buildings are at present completed for the accommodation of six hundred patients, and additional buildings for four hundred or five hundred are now under construction. All classes of epileptics not insane, above the age of seven or eight years, will be admitted. The institution is supported exclusively by the State, and the appropriation for the coming two years is at the rate of $190 per capita. Twenty-five dollars per year is allowed for clothes, to be paid by the friends or relatives of the patient or by the township from which he comes. Patients are committed through the Director of the Poor of their district, but Judges of the County Courts may commit vagrant or feeble-minded individuals at State cost. Pennsylvania has an epileptic population of from fourteen to sixteen thousand authenticated cases. The opinion is stated that under proper institutional management, from eleven per cent to seventeen per cent of epileptics can be cured; treatment in the home or outside of an institution, from two per cent to three per cent. Schools will be maintained. There will be two physicians in addition to the superintendent. A laboratory and training school will be maintained.

The title of this institution indicates that it is for the accommodation of feeble-minded as well as of epileptics. It is purposed at a comparatively early date to introduce a bill into the Legislature making this institution solely for the care and treatment of epileptics.

DR. FITZSIMMONS.

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PENNSYLVANIA EPILEPTIC HOSPITAL AND COLONY FARM AT OAKBOURNE, PA.

DR. WHARTON SINKLER stated:

That this institution was started by private subscription and largely by the liberality of a few individuals. The farm, consisting of 114 acres, was given by two persons, and a gentleman of great benéficence supplied the means for erecting all of the buildings. The institution may be called a semi-state institution, as we receive support from the State amounting only to about one-quarter of what it costs to maintain the institution. We have two cottages, one for men and one for women, each of which accommodates twenty-nine patients. What I wish especially to call your attention to, is the fact that we are now erecting and have almost finished a cottage for children which will accommodate thirty-two children and which will cost about $23,000. In this building we propose to have school rooms and rooms for manual training, Sloyd work and other work of this kind. It is certainly a matter of great importance to have a department devoted exclusively to children. If we can admit children before the disease fully develops, there is a much better chance of effecting a cure. I am afraid that the proportion of cures which we have had will not compare favorably with the figures which have been given by several other institutions. It is necessary, however, to determine what constitutes a cure. If freedom from fits for one or two years constitutes a cure, then we have had several. A number of our patients have had no fits for two years and these have been sent home and we have heard nothing further from them. It seems to me, however, that we should consider freedom from convulsions for at least five years necessary to constitute a cure.

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The State of Pennsylvania has begun the erection of a large institution for the Feeble-Minded and Epileptics. The institution is now ready to receive about 500 patients, and it is expected when completed that it will accommodate about 1,800. The question is, whether an institution like this will interfere with our small institution at Oakbourne. I believe not, because of the peculiar class of patients whom we admit. We admit none who are insane or incapacitated in any way for employment. All whom we admit are high grade epileptics and are capable of employment of some kind, and therefore can help to support themselves.

TEXAS

The State Epileptic Colony is located at Abilene, Texas. It was founded in 1903. Its population October 1, 1906, was 260. All classes of epileptics are admitted above eight years of age, except idiots and imbeciles. Patients are supported by the State, upon commission by the County Court. Private or pay patients are also received, The per capita cost for the last fiscal year was $199. The principal industries were farming and gardening, and their estimated value was $11,072. The estimated epileptic population of Texas is 3,000 and the probable size to which the Colony will grow is 1,000. Buildings are now being erected for 100 cases. There is one physician on the staff beside the Superintendent. No laboratory or training school for nurses or schools for patients are maintained.

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The wages for male attendants is given at $20 per month and for women the same; special nurses $30 per month. The curability of epilepsy is placed at five per cent.

Reported by Dr. John Preston.

THE MASSACHUSETTS HOSPITAL FOR EPILEPTICS

The Massachusetts Hospital for Epileptics is located at Palmer, Mass. It was founded in 1895 and is a combined colony and hospital. Its capacity on October 1, 1906, was 700 and the institution will probably not exceed a capacity of 1,000, including cases of school age, although the estimated epileptic population of Massachusetts is 10,000. Sane and insane epileptics of all grades of intelligence, above the age of ten years, are received as patients, and are classified in separate cottages. Patients are maintained at state, town, and private expense, the per capita cost for the last fiscal year being $223, ($4.30 per week.) Patients are committed by any court or upon personal application if able to pay $5 per week.

A table showing the employment of patients indicates that housework, laundry-work, sewing and farming are the principal occupations, while the trades are also represented. The value of patients' labor, estimating men at twenty cents and women at ten cents per day, was $15,180 and $6,930 respectively, a total of $22,110 for the fiscal year. Five per cent of the patients attend schools maintained by the Colony. The medical staff numbers four beside the Superintendent, and a laboratory is maintained.

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Regarding the curability of epilepsy, out of all cases fifty per cent ceased to have fits; of established cases two to twenty per cent ceased to have fits; of very old cases from one per cent to five per cent ceased to have fits.

Men attendants are paid from $25 to $55 per month, and women from $20 to $40.

A training school with a three years' course was established in 1900.

Reported by Dr. Flood.

DR. BULLARD OF MASSACHUSETTS:

A law has been passed this year enabling us to take children at the age of eleven years, while previously the law was such that we could not take them until they were fourteen. This will make a considerable difference in the opportunities of the institution and possibly enable us to obtain better results. I hope the time will come when all the epileptic children can be received by us.

NEW YORK

The most important event in this State during the last year, was the creation of a Commission to select a site for a new institution in the southeastern part of the State. This institution is primarily designed to take low grade epileptics and other custodial cases.

At Sonyea, there had been 2,262 admissions since the beginning, with a remaining population of 1,081 on October 1, 1907. Two new cottages for men are partly occupied, and one for women is also practically complete and in

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partial use. Two more cottages for women will be opened in the near future; these buildings will give room for about 200 additional colonists.

The net cost of maintenance for the past year was $173.4059 per colonist. The value of the industries was $54,817.55.

The staff is composed of the Superintendent and seven other physicians. A training school is conducted by members of the staff.

The Colony possesses a well equipped Laboratory in charge of a Pathologist. Material of all kinds is available in ample amount. Chapter 458, Laws 1905, gives the Colony the right to remove the brain from certain indigent patients dying at the Colony. This, with autopsies obtained in the ordinary way gave fifty-one brains out of a total of seventy-two deaths during the past year. The study of dead material, however, has been put aside for the present, in favor of the study of the living epileptic. It has been decided more especially to study those epilepsies assumed to arise from abnormal conditions of the digestive tract: the methods of investigation will necessarily be chemical and bacteriological, and we hope in course of time to be able to report some facts of interest if not of importance.

The separating for purposes of study of cases apparently similar is, for us at least, a step in advance. When first taking up the work, the correct method of approach seemed to be the studying of large groups of cases. It has been impressed on us, however, that the epileptic cannot be considered en masse but only as an individual. We are dealing with epilepsies, and not with epilepsy.

W. P. SPRATLING.

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REPORT FROM GEORGIA

There is no institution especially for epileptics in Georgia. Insane epileptics are cared for at the Georgia State Sanatorium at Milledgeville. Epileptics who commit crimes are sent to prison. Some are cared for in work houses or on farms. The total number of epileptics in Georgia is estimated at 2,500. Georgia is agitating at this time the advisability of establishing a Colony or Village for the especial care of this class.

VIRGINIA

The first suggestions and efforts in the matter of a Colony, or separate State institution, for epileptics in Virginia were made in 1894, and since then the matter has been kept constantly before the public. Now it has universal support throughout the State. The Governor and Board of Directors of the State Hospitals for the Insane have given the proposition their hearty cooperation. The last Legislature made a small appropriation for the establishment of a Colony for insane white epileptics, to be conducted in connection with one of the Insane State Hospitals. This Hospital had donated to it a picturesque site on the James River, near Lynchburg, in the far-famed and beautiful Piedmont section, on condition that an institution of its character be established thereon. Progress is being made in securing plans and specifications for suitable buildings for about one hundred patients, who will be removed from the State Hospitals for the Insane.

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The Colony will start on a rather small scale, and its sphere of usefulness will necessarily be limited, yet it will doubtless develop into a great separate institution for both sane and insane epileptics. The next Legislature will probably appropriate sufficient money to enlarge the Colony, to properly equip it, and to place it upon a firm basis of usefulness.

There are 3,000 epileptics in the State and 370 in Hospitals.

Reported by Dr. W. F. Drewry.

NEW JERSEY

The New Jersey State Village for Epileptics, located at Skillman, was founded November 1, 1898, and had on October 1st an epileptic population of 200. All epileptics over five years of age are received, except the idiotic and insane. Indigent cases are committed by the Court of Common Pleas. The institution is supported by the State and counties, at a per capita cost of $217 per year. The chief industry is agriculture. There are about 3,000 epileptics in the State and it is stated that about seven per cent of all cases are curable.

About thirty-five children attend school. There are two physicians on the staff beside the Superintendent. The wages paid male attendants are from $18 to $28 and women $16 to $25. No training school is maintained.

Reported by Dr. H. M. Weeks.

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CALIFORNIA

A movement was inaugurated in the southern part of the State last November looking toward public care of epileptics.

ILLINOIS

William C. Graves, Executive Officer of the Illinois State Board of Charities, reported for the State of Illinois as follows:

"There has been a law on the statute books of the State of Illinois since 1899 creating the Illinois Colony for Epileptics. This law contemplates the creation of a separate colony for epileptics, with buildings on the cottage plan. The legislature did not appropriate for the actual erection of the institution, but for preliminary steps such as selecting and securing an option on a site and preparing plans and specifications for buildings and equipment. Owing to various causes, efforts to secure a sufficient appropriation to erect and equip an institution have failed.

"Illinois just now is in a great epoch of advancement in public charity administration. The present State Board of Charities, headed by Dr. Frank Billings, conducted a campaign in the legislature last winter for the rehabilitation of the physical property and for modernizing the service of the seventeen state charitable institutions, particularly those in the group devoted to the insane and the feeble-minded. It also recommended and worked for an appropriation of $265,000 to purchase not fewer than 1,000 acres of land and to establish thereon the nucleus (capacity

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of 200 colonists) of an institution for improvable epileptics. As a part of this campaign Dr. William P. Spratling of Sonyea, New York, came to Springfield and delivered an illustrated lecture on epilepsy and epileptics. The Board of Charities made a careful canvas of the epileptics, sane and insane, in all state institutions (except the two prisons and reformatory) and in county almshouses. The following table shows the result of this canvas.

Epileptics in State institutions	1015
Epileptics in County almshouses	160
Total	1175

"Of the 1,015 in state institutions, 561 were inmates of hospitals for the insane and the remaining 454 were in the institution for feeble-minded and in other charitable institutions. It is estimated that there are 10,000 epileptics in Illinois in and out of institutions. Dr. F. S. Churchill, President of the Children's Hospital Society of Chicago, prepared the general literature used in the campaign before the people and the legislature. Dr. Hugh T. Patrick of Chicago and others also rendered valuable assistance.

"In response to the efforts of the State Board of Charities the legislature appropriated $1,500,000 more for charitable purposes than it did two years ago, and it also appropriated $500,000 toward a new $3,000,000 penitentiary, recommended by the Board of Charities.

"In view of the large additional sum voted for existing institutions and for the new penitentiary, the legislature felt itself unable to appropriate for the epileptic colony or for a state sanatorium for consumptives.

"I firmly believe that within the very near future our legislature, which has already created an epileptic colony

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on paper, will provide the necessary funds to build the institution. The educational work has been done in a large measure. I believe only the finishing touches and a more financially favorable time are required to give us the new institution which we need so much. You think the outlook is favorable in Illinois, don't you, Dr. Brower?''

Dr. Daniel R. Brower of Chicago: ''Yes, Mr. Graves, I agree with you entirely.''

REPORT FROM OHIO

By William H. Pritchard, M. D.
Superintendent of the Ohio Hospital for Epileptics at Gallipolis.

There have been under treatment at the Ohio Colony during the fiscal year ending November 15th last, a total of 1,639 patients of whom 923 were males and 716 were females. There were on the register on that day 745 males and 607 females, 1,352 in all. The admission during the year numbered 168 males and 102 females, a total of 270. The daily average of patients actually residing in the institution during the year was 1,294. There were forty-six patients absent on furlough at the close of the year. The per capita cost for total maintenance including expenditures for all purposes other than permanent repairs and improvement was $173.66. The net cost to the State for maintenance was $155.88 per patient. For permanent improvements including repairs to existing buildings, extensive alterations and additions to a substantial brick residence fitting it for occupancy as a cottage, have been made and a large amount of grading and farm improve-

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ments have been completed. A small parcel of land adjoining the institution grounds has been purchased.

It was stated in my report published in the Transactions last year that the institution owned over five hundred acres of land. This was a mistake, the actual amount being only four hundred and fifty-one acres. Appropriations for purchasing about fifty acres more have been requested from the Legislature which meets in January, 1908. Requests for about $125,000.00 for repairs and new construction have also been made, the principal items included therein being a cottage for women patients, an assembly and amusement hall, a farm and dairy barn, a greenhouse, and additional boiler room, electrical, and laundry equipment. There will be required for maintenance during the present year about $216,000.00.

The government of the institution is vested in a board of five trustees who are appointed by the Governor and who serve for terms of five years each. Trustees receive no pay except their necessary expenses. They meet at the hospital once each month for the purpose of auditing accounts and making a general inspection of the institution. The trustees appoint the Superintendent and the resident medical and fiduciary officers. All other employes are appointed by the Superintendent.

As at present constituted the Colony comprises thirty-six buildings, exclusive of greenhouses, barns, and outhouses. Of these thirteen are occupied by male patients, eight by female patients, and two, the general hospital and the custodial building for insane epileptics, by patients of both sexes. Eight of the cottages occupied by male patients are now in charge of man and wife as attendants. In three of these the woman does the cooking and superintends the housekeeping in general. In the other five she

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does the ordinary inside work of an attendant. By this innovation the housekeeping and the care given to patients' clothing has been much improved. At the institution hospital women have been substituted for men in the care of male patients with excellent results. As opportunity offers the plan will probably be extended. I have serious doubts, however, of the advisability of placing women in charge of violently insane epileptic patients.

The routine medical work of the Colony is in charge of five assistant physicians, all of whom have had from two to twelve years' practical experience in institution work. All newly admitted patients who are not violently insane are sent first to the Colony hospital where they are given a thorough physical and mental examination and the findings recorded. They are then assigned to a cottage befitting their condition and come directly under the daily observation of the physician in charge. In connection with the hospital the Colony has an excellently equipped laboratory and a large amount of clinical laboratory work is being constantly done. It is a matter of constant regret that for several years past the state has failed to provide funds with which to secure the services of a competent pathologist, and it has therefore been impossible to regularly prosecute original research work. Desultory studies along certain lines have been pursued with some show of results, but the services of a trained physiological chemist and pathologist who can devote his entire time to the work is greatly to be desired. It is hoped that the Legislature will change the present statutes in such a way as will render possible the employment of such a person.

In the fourteen years of its existence the Colony has had under its care a total of 3,649 epileptic patients of all classes. Of these there have been discharged as recovered

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258, a percentage of seven and three-tenths. Many of these have later suffered a recurrence and have been returned. The actual percentage of recoveries, therefore, has probably not been over five. There were ten so discharged during the past year. There can be no reasonable doubt of the curability of epilepsy in many cases when the first manifestations of the disease are delayed until between the ages of twelve and twenty years, and when colony treatment is instituted promptly after the malady is first recognized. The most serious obstacle to the successful management of epileptics is the reluctance with which both patients and friends consent to the commitment of the patient to an institution. The greatest boon to the epileptic would be compulsory commitment to a colony as soon as the disease has become established. Of 3,649 patients admitted into the Ohio institution in fourteen years only 138 have come in during the first year of the existence of the malady; in 925 cases the disease had existed for less than five years, while in 2,724 cases the duration previous to admission had been more than five years.

Efforts have been constantly made to give increased prominence to the educational, amusement, and employment features of colony life. Seventy-six boys and girls between the ages of seven and nineteen are enrolled in day school, and increasing numbers of young and intelligent patients are being assigned to work at the various handicrafts under the direction of mechanics in charge of the shops. Young men are given regular employment in the bakery, kitchen, butcher shop, carpenter shop, and tin shop, and at upholstering, painting, plumbing and printing. Girls and women are extensively employed in cooking, sewing, laundry work and housework. The training

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they get in these departments is practical. Many of them have already gone through the day school. Older patients of the intermediate class are usually given employment at outdoor work, such as grading and the care of the lawns and shrubbery. Special details are assigned to the farm and gardens. About sixty per cent of all patients are assigned some regular work. The usual forms of amusement and diversion have been provided in abundance. Particular attention has been paid to outdoor sports, and successful baseball, football, and basket ball teams have been maintained. An amateur theatrical club has been organized and frequent performances by traveling companies have been given. Proper social intercourse between patients of the better class has been encouraged and a movement to establish a circulating library and club rooms for both sexes is under way.

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REPORT OF THE NOMINATING COMMITTEE

Mr. President and Members of the Association:

Your nominating committee reports as follows:

For President, Dr. H. M. Weeks, Superintendent of the New Jersey State Village for Epileptics.

For 1st Vice-President, Dr. W. F. Drewry, Superintendent of Central State Hospital, Petersburg, Va.

For 2d Vice-President, Dr. Thomas C. Fitzsimmons, member of the Commission to build the new Institution for Epileptics in Pennsylvania.

For Secretary and Treasurer, Dr. J. F. Munson, of The Craig Colony for Epileptics, Sonyea, N. Y.

The above are all the offices provided for in the Constitution.

DR. W. N. BULLARD, of Mass.,
Chairman.

The above report was adopted by the Association,

J. F. MUNSON,
Secretary.

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ORGANIZATION OF THE ASSOCIATION
FOR 1908

PRESIDENT:
DR. H. M. WEEKS,
SKILLMAN, N. J.

FIRST VICE-PRESIDENT:
DR. W. F. DREWRY,
PETERSBURG, VA.

SECOND VICE-PRESIDENT:
DR. T. C. FITZSIMMONS,
WILKESBARRE, PA.

SECRETARY AND TREASURER:
DR. J. F. MUNSON,
SONYEA, N. Y.

EXECUTIVE COMMITTEE:
DR. W. T. SHANAHAN, *Chairman*,
NEW YORK.

DR. W. N. BULLARD,
MASSACHUSETTS.

DR. ADOLPH MEYER,
NEW YORK.

DR. E. E. SOUTHARD,
MASSACHUSETTS.

MR. W. C. GRAVES,
ILLINOIS.

DR. L. PIERCE CLARK,
NEW YORK

DR. W. H. PRITCHARD,
OHIO.

DR. W. O. VAN NUYS,
INDIANA.

DR. EVERETT FLOOD,
MASSACHUSETTS.

DR. J. ALLISON HODGES,
VIRGINIA.

DR. WILLIAM P. SPRATLING,
SONYEA, N. Y.
Permanent Editor Transactions; Chairman Committee on Membership.

OTHER MEMBERS OF COMMITTEE ON MEMBERSHIP:
DR. EVERETT FLOOD,
PALMER, MASS.

DR. L. PIERCE CLARK,
23 W. 58TH ST., NEW YORK CITY.

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COMPOSITION OF THE MEMBERSHIP OF THE NATIONAL ASSOCIATION FOR THE STUDY OF EPILEPSY AND THE CARE AND TREATMENT OF EPILEPTICS

The membership of the Association is as follows:

1. ACTIVE MEMBERS pay annual dues of $5.00, are entitled to attend all meetings of the Association, take part in all discussions and receive the Annual Transactions handsomely bound free of additional cost.

2. ASSOCIATE MEMBERS pay annual dues of $3.00, may attend the meetings of the Association and take part in the discussions. Associate members receive the annual Transactions of the Association. They cannot vote or hold office.

3. HONORARY AND CORRESPONDING MEMBERS are exempt from dues of any kind. They are entitled to attend all meetings of the Association; take part in all discussions and receive free the Annual Transactions.

If you desire your name to appear as an active member in the future, please send $5.00 for your yearly dues for 1908 to Dr. James F. Munson, Secretary-Treasurer, Sonyea, Livingston County, New York.

William P. Spratling, M. D., *Chairman.*
L. Pierce Clark, M. D., Everett Flood, M. D.
Committee on Membership.

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MEETING OF THE EXECUTIVE COMMITTEE

9:30 P. M., Oct. 23d,

AT THE JEFFERSON HOTEL, RICHMOND, VA.

Present, Dr. Drewry, Chairman, Dr. Flood, Dr. Fitzsimmons, Dr. Bullard, Dr. Shanahan, Dr. Munson, Secretary.

Dr. Brower of Chicago and Mr. Graves of Springfield, Ill., were also present.

The following communication and letter were read from Dr. Adolf Meyer:

Doctor Everett Flood,
Hotel Jefferson,
Richmond, Va.

My dear Doctor:

To my great regret I am unable to come to the meeting. I have, however, prepared a statement concerning the propositions of which we spoke at our Washington conference.

Doctor Clark suggests that the Association should empower or guarantee the payment of not less than $200 a year for the abstracting and editing of the report; and also that a committee of three be appointed to supervise the work.

With my best wishes for the success of the meeting, I remain.

Faithfully yours,
ADOLF MEYER.

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SUGGESTIONS CONCERNING AN ANNUAL REPORT OF THE WORK DONE IN THE INVESTIGATION OF THE EPILEPTIC

By Dr. Adolf Meyer

In a field so complex as that of epilepsy the physicians live in institutions remote from medical centers, and even those near such centers must frequently feel the difficulty in finding in such a wealth of literature what is essential, and of sifting material so as to make plans for their own work. This strongly felt fact receives its practical illustrations in the frequently disjointed returns of well-intended work done by those not closely enough in touch with the most profitable helps. Suggestions of various kinds have been made to overcome the difficulties and to see that the serious problem of the nature and prevention of epilepsy should be attacked more profitably.

In harmony with the trend of general centralization, central laboratories have been thought of, but the very reasonable objection has been made that one small group of workers cannot expect to do everything, and that what is most wanted is helps in doing the work as well as possible wherever there are patients under the care of physicians and wherever there are opportunities for observation of facts and their interpretation for purposes of action; and this refers to the study of the clinical as well as the anatomical issues. It is a well-recognized fact that no hospital can now-a-days get along without an adequately

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equipped clinical and anatomical laboratory. It is also plain that every individual worker can only expect to cover a relatively small field, and it becomes desirable to in some way connect these scattered labors.

It is, therefore, suggested that as far as possible an annual account of the work performed on the problem of epilepsy and related facts be given in connection with the Annual Transactions of the Association. Such an annual survey is, of course, a very difficult problem and presupposes excellent bibliographical and library facilities, and what is much more rare, a capacity for digesting the results in a more or less critical manner. It is, however, to be hoped that such an undertaking will bring into the hands of the editorial staff of the Annual Transactions a great deal of valuable information, reprints, and assistance prompted by the desire of obtaining help for definite pieces of research, for indeed the central committee may ultimately be looked upon as the distributor of funds such as are not already obtainable from the local authorities.

In connection with the review of more strictly scientific facts, the annual report should include a summary of reports sent by foreign correspondents on the movements of the practical problem of the care of epilepsy in the various countries.

This project brings with it very serious responsibilities, but it will undoubtedly stimulate many physicians to take a broad view of what is being done and to lend a hand in the annual preparation of a work which will surely be greatly to the credit of the Association.

After discussion upon Dr. Meyer's communications, it was moved and carried that the report of Dr. Meyer be

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accepted with the following modification: That a committee of three be appointed by the President to make a yearly summary of work performed throughout the world upon the problem of epilepsy and related points, this report to be published in the Annual Transactions of the Association. It shall further be the duty of this committee to suggest special work for each group of workers *if requested.*

It was moved and carried that the out-going President appoint this committee.

Dr. Flood, President of the Association, named Drs. Clark, Meyer and Southard as this committee.

The suggestion of Dr. Meyer relative to guaranteeing $200 was discussed but no action was taken. Dr. Drewry was called out at this time and Dr. Flood took the chair.

It was moved and carried that "Corresponding Members" be added to the third class members, as suggested in the transactions of the New York meeting of the Executive Committee.

It was the sense of the meeting that the Chairman of the Executive Committee act as Press Agent.

It was the sense of the meeting that those names on the list of members from whom no response has been received should be dropped from the roll.

It was moved, seconded and carried that the fiscal year end May 31st.

The meeting then adjourned.

J. F. MUNSON,
Secretary and Treasurer.

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CONSTITUTION

Article I

The name of the Association shall be "The National Association for the Study of Epilepsy and the Care and Treatment of Epileptics."

Article II

The objects of the Association shall be:

1. To promote the general welfare of sufferers from epilepsy.
2. To stimulate the study of the causes and methods of cure of this disease.
3. To assist the various States in America in establishing a proper system of care for epileptics.
4. To advocate the care of epileptics in institutions designed to meet their special needs.

Article III

Any person interested in the scientific study of epilepsy, or in the study of the means that aim to aid the epileptic, or in social science matters generally, expressing a desire to become a member of the Association, may be recorded as such with the approval of the Executive Committee.

Article IV

The officers of the Association shall consist of a President, a First Vice-President, a Second Vice-President, a Secretary and a Treasurer. They shall be elected annually by the members present at annual meeting of the Association. In case of death or resignation of any officer his remaining term shall be filled by a member elected by the Executive Committee.

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ARTICLE V

The President shall preside at all meetings of the Association or appoint a presiding officer from those officers who are present. He shall appoint the members of standing and special committees, and shall have the objects and general interests of the Association in charge.

ARTICLE VI

The Secretary shall keep minutes of all meetings of the Association. He shall give notice when directed by the Executive Committee of all special meetings and keep such accounts and records as may be required.

ARTICLE VII

The Treasurer shall collect and have charge of the funds of the Association, shall keep a regular account thereof, and make reports thereof to the Executive Committee as required by the Committee, and an annual report to the Association. Claims against the Association shall be paid by the Treasurer only upon the audit of the President of the Association and the Chairman of the Executive Committee.

ARTICLE VIII

There shall be an Executive Committee composed of such members of the Association as may be appointed by the President. The President shall be, ex-officio, a member of this Committee. It shall be the duty of this Committee to define and adopt such course of action as may best promote the objects of the Association and to devise ways and means for increasing the usefulness and efficiency of the Association.

ARTICLE IX

This constitution may be amended by a two-thirds vote of the members present at the annual meeting, thirty days' previous notice having been given in writing of such proposed change by the Secretary to all members of the Association.

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HONORARY MEMBERS

HON. WM. PRYOR LETCHWORTH, LL. D., Glen Iris, P. O. Portage, N. Y.

PROF. WILLIAM OSLER, Oxford University, England.

DR. HUGHLINGS JACKSON, No. 3 Manchester Square, London, England.

MISS LOUISE LEE SCHUYLER, Gramercy Park, New York City.

MISS JULIA LATHROP. Commiseioner State Board of Charities, Rockford, Illinois.

MRS. ANDREW H. SMITH, Geneva, N. Y.

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LIST OF ACTIVE MEMBERS OF THE NATIONAL ASSOCIATION FOR THE STUDY OF EPILEPSY, THE CARE AND TREATMENT OF EPILEPTICS

DR. E. B. ANGELL, Rochester, N. Y.
DR. B. F. ANDREWS, Sonyea, N. Y.
DR. S. T. ARMSTRONG, 144 E. 37th St., New York City.
DR. MARY L. AUSTIN, Gallipolis, O.
DR. PEARCE BAILEY, 52 W. 53rd St., New York City.
DR. C. C. BELING, Newark, N. J.
DR. W. N. BULLARD, 89 Marlborough St., Boston, Mass.
MRS. M. R. BULLARD, 89 Marlborough St., Boston, Mass.
MRS. W. S. BULLARD, 3 Commonwealth Ave., Boston, Mass.
MR. H. E. BROWN, Mt. Morris, N. Y.
MR. AMOS W. BUTLER, Indianapolis, Ind.
DR. C. B. BURR, Flint, Mich.
DR. DANIEL R. BROWER, 34 Washington St., Chicago, Ill.
DR. WALTER G. CHASE, 45 Beacon St., Boston, Mass.
DR. G. L. CHAMBERLAIN, Lapeer, Mich.
DR. J. W. CHAMBERS, 18 Franklin St., Baltimore, Md.
DR. L. PIERCE CLARK, 23 W. 58th St., New York City.
MR. CHARLES A. CLOUGH, Groton, Mass.
MR. T. A. COGHLAN, 123 Cannon St., London, Eng.
DR. G. KIRBY COLLIER, Sonyea, N. Y.
DR. H. A. COTTON, Trenton, N. J.
DR. EDWARD COWLES, 419 Boylston St., Boston, Mass.
DR. HARVEY CUSHING, Baltimore, Md.
DR. OWEN COPP, Room 36, State House, Boston, Mass.
DR. FLOYD M. CRANDALL, 113 W. 95th St., New York City.
MR. ALLAN D. CONOVER, Madison, Wis.
DR. CHARLES L. DANA, 53 W. 53d St., New York City.
MRS. JOHN DAVENPORT, Bath, N. Y.
MR. C. C. DESMOND, 301 S. Spring St., Los Angeles, Cal.
MR. ABBOTT L. DOW, Brooklyn, N. Y.
DR. WM. E. DOLD, The Sydenham, 616 Madison Ave., New York City.
DR. E. A. DOWN, Hartford, Conn.
DR. W. F. DREWRY, Petersburg, Va.

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DR. G. E. EPLER, 703½ Garrison Ave., Fort Smith, Ark.
DR. HENRY L. ELSNER, Syracuse, N. Y.
DR. B. D. EVANS, Morris Plains, N. J.
DR. W. N. FERNALD, Waverly, Mass.
DR. J. G. FISHBOURNE, 80 Collins St., Melbourne, Australia.
DR. THOMAS C. FITZSIMMONS, Carbondale, Pa.
DR. EVERETT FLOOD, Palmer, Mass.
DR. W. A. GORDON, Winnebago, Wis.
MR. D. M. GARSON, Rochester, N. Y.
DR. MORGAN B. HODSKINS, Palmer, Mass.
DR. GEO. E. GORHAM, 214 State St., Albany, N. Y.
DR. F. K. HALLOCK, Cromwell, Conn.
DR. CHRISTIAN A. HERTER, 819 Madison Ave., New York City.
DR. S. S. HILL, Wernersville, Pa.
DR. J. ALLISON HODGES, Richmond, Va.
DR. E. H. HOWARD, Rochester, N. Y.
MR. W. C. GRAVES, Springfield, Ill.
DR. HUGHLINGS JACKSON, Manchester Square, London, Eng.
DR. H. B. JACOBS, 11 W. Mt. Vernon Place, Baltimore, Md.
DR. SMITH ELY JELLIFFE, 64 W. 56th St., New York City.
PROF. E. R. JOHNSON, Vineland, N. J.
DR. W. ERNEST JONES, Victoria, Australia.
DR. ABRAHAM JACOBI, 19 E. 47th St., New York City.
MR. CHARLES P. KELLOGG, Waterbury, Conn.
DR. E. A. KENNEDY, Palmer, Mass.
DR. FRANK W. KEATING, Owings Mills, Md.
MR. F. B. KIRKBRIDE, 37 Madison Ave., New York City.
DR. GEORGE H. KNIGHT, Lakeville, Conn.
DR. W. C. KRAUSS, 371 Delaware Ave., Buffalo, N. Y.
DR. E. LAVAL, Blackburn, Eng.
MR. C. M. LEA, Philadelphia, Pa.
DR. MAX MAILHOUSE, 45 Elm St., New Haven, Conn.
DR. EDWARD E. MAYER, 524 Penn. Ave., Pittsburg, Pa.
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